

=> s amyloid(w)beta(w)peptide  
L1 12537 AMYLOID(W) BETA(W) PEPTIDE  
=> s beta(w)amyloid(w)peptide  
L2 9399 BETA(W) AMYLOID(W) PEPTIDE  
=> s nogo(w)receptor(w)antagonist  
L3 13 NOGO(W) RECEPTOR(W) ANTAGONIST  
=> d 13 1-13

L3 ANSWER 1 OF 13 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN  
AN 2006:436251 BIOSIS  
DN 2006:436251 BIOSIS  
TI Nogo goes in the pure water: Solution structure of Nogo-60 and design of  
the structured and buffer-soluble Nogo-54 for enhancing CNS regeneration.  
AU Li, Minfen; Liu, Jingxian; Song, Jianxing [Reprint Author]  
CS Natl Univ Singapore, Dept Biochem, Yong Loo Lin Sch Med, 10 Kent Ridge  
Crescent, Singapore 119260, Singapore  
bchsjen@nus.edu.sg  
SO Protein Science, (AUG 2006) Vol. 15, No. 8, pp. 1835-1841.  
ISSN: 0961-8368.  
DT Article  
LA English  
ED Last Updated on STN: 30 Aug 2006

L3 ANSWER 2 OF 13 CAPLUS COPYRIGHT 2007 ACS on STN  
AN 2006:1226364 CAPLUS  
DN 146:26348  
TI Neuronal degeneration treatment with Nogo receptor  
antagonists  
IN Lee, Daniel H. S.; Sah, Dinah W. Y.; So, Kwok Fai; Wu, Wutian  
PA Biogen Idec MA Inc., USA; The University of Hong Kong  
SO PCT Int. Appl., 49pp.  
CODEN: PIXXD2  
DT Patent  
LA English  
FAN.CNT 1

PI WO 2006124627 A2 20061123 WO 2006-US18484 20060512  
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,  
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VN, YU, ZA, ZM, ZW  
KG, KZ, MD, RU, TJ, TM  
P 20050512  
PRAI US 2005-67995P P 20051110  
US 2005-735187P

L3 ANSWER 3 OF 13 CAPLUS COPYRIGHT 2007 ACS on STN  
AN 2006:289503 CAPLUS  
DN 145:241471  
TI Delayed intraventricular Nogo receptor  
antagonist promotes recovery from stroke by enhancing axonal  
plasticity

AU Kim, Tae-Mon; Lee, Jung-Kil; Joo, Sung-Pil; Kim, Tae-Sun; Kim, Jae-Hyoo;  
CS Kim, Soo-Han  
Department of Neurosurgery, Chonnam National University Hospital, Gwangju,  
S. Korea  
SO Journal of Korean Neurosurgical Society (2006), 39(2), 130-135  
CODEN: JKNSAI.  
PB Korean Neurosurgical Society  
DT Journal  
LA English  
RE.CNT 25  
THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 4 OF 13 CAPLUS COPYRIGHT 2007 ACS on STN  
AN 2005:823596 CAPLUS  
DN 143:222540  
TI Treatment of conditions involving dopaminergic neuronal degeneration using  
Nogo receptor antagonists  
IN Reiton, Jane K.; Engber, Thomas M.; Strittmatter, Stephen M.  
PA Biogen Idec MA Inc., USA; Yale University  
SO PCT Int. Appl., 26 pp.  
CODEN: PIXXD2  
DT Patent  
LA English  
FAN.CNT 1

PI WO 2005074972 A2 20050818 WO 2005-US2535 20050128  
WO 2005074972 A3 20051222  
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,  
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PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, TJ, TM, TN, TR, TT, TZ,  
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VN, YU, ZA, ZM, ZW  
MR, NE, SN, TD, TG  
AU 2005210621 A1 20050818 AU 2005-210621 20050128  
CA 2555018 A1 20050818 CA 2005-2555018 20050128  
EP 1713494 A2 20061025 EP 2005-712127 20050128  
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE,  
SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, BA, HR, IS, YU  
CN 1946418 A 20070411 CN 2005-80009242 20050128  
PRAI US 2004-540798P P 20040130  
WO 2005-US2535 W 20050128

L3 ANSWER 5 OF 13 CAPLUS COPYRIGHT 2007 ACS on STN  
AN 2005:564795 CAPLUS  
DN 143:91068  
TI Methods of stimulating axonal growth of CNS neurons using Nogo  
receptor antagonists in combination with growth factors  
IN Benowitz, Larry I.; Fischer, Dietmar  
PA Children's Medical Center Corporation, USA  
SO PCT Int. Appl., 74 pp.  
CODEN: PIXXD2  
DT Patent  
LA English  
FAN.CNT 1

PI WO 2005059515 A2 20050630 WO 2004-US42255 20041216

[illegible]

DN 139:332941  
 TI Delayed systemic Nogo-66 receptor antagonist promotes recovery from spinal cord injury  
 AU Li, Shuxin; Strittmatter, Stephen M.  
 CS Department of Neurology and Section of Neurobiology, Yale University School of Medicine, New Haven, CT, 06520, USA  
 SO Journal of Neuroscience (2003), 23(10), 4219-4227  
 CODEN: JNRSDS; ISSN: 0270-6474  
 PB Society for Neuroscience  
 DT Journal  
 LA English  
 SL English  
 RE.CNT 50  
 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD  
 ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 9 OF 13 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN  
 AN 2006363205 EMBASE  
 TI Nogo goes in the pure water: Solution structure of Nogo-60 and design of the structured and buffer-soluble Nogo-54 for enhancing CNS regeneration.  
 AU Li M.; Liu J.; Song J.  
 CS J. Song, Department of Biochemistry, Yong Loo Lin School of Medicine, National University of Singapore, 10 Kent Ridge Crescent, Singapore 119260, Singapore. bchs@nus.edu.sg  
 SO Protein Science, (2006) Vol. 15, No. 8, pp. 1835-1841.  
 Refs: 30  
 ISSN: 0961-8368 E-ISSN: 1469-896X CODEN: PRCEBI  
 CY United States  
 DT Journal; Article  
 FS 029 Clinical Biochemistry  
 LA English  
 SL English  
 ED Entered STN: 18 Aug 2006  
 Last Updated on STN: 18 Aug 2006

L3 ANSWER 10 OF 13 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN  
 AN 2005100947 EMBASE  
 TI The newt in us.  
 AU Rinaldi A.  
 SO EMBO Reports, (2005) Vol. 6, No. 2, pp. 113-115.  
 Refs: 10  
 ISSN: 1469-221X CODEN: ERMEAX  
 CY United Kingdom  
 DT Journal; Article  
 FS 008 Neurology and Neurosurgery  
 021 Developmental Biology and Teratology  
 029 Clinical Biochemistry  
 030 Pharmacology  
 037 Drug Literature Index  
 LA English  
 SL English  
 ED Entered STN: 17 Mar 2005  
 Last Updated on STN: 17 Mar 2005

L3 ANSWER 11 OF 13 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN  
 AN 2003476128 EMBASE  
 TI Signaling mechanisms of the myelin inhibitors of axon regeneration.  
 AU Yiu G.; He Z.  
 CS Z. He, Division of Neuroscience, Children's Hospital, Harvard Medical School, Boston, MA 02115, United States. zhiang.he@ch.harvard.edu  
 SO Current Opinion in Neurobiology, (2003) Vol. 13, No. 5, pp. 545-551.  
 Refs: 67  
 ISSN: 0959-4388 CODEN: COPUEN

CY United Kingdom  
 DT Journal; General Review  
 FS 008 Neurology and Neurosurgery  
 030 Pharmacology  
 037 Drug Literature Index  
 LA English  
 SL English  
 ED Entered STN: 4 Dec 2003  
 Last Updated on STN: 4 Dec 2003

L3 ANSWER 12 OF 13 TOXCENTER COPYRIGHT 2007 ACS on STN  
 AN 2006386140 TOXCENTER  
 CP Copyright 2007 ACS  
 DN CA14602026348C  
 TI Neuronal degeneration treatment with Nogo receptor antagonists  
 AU Lee, Daniel H. S.; Sah, Dinah W. Y.; So, Kwok Fai; Wu, Wutian  
 CS ASSIGNEE: The University of Hong Kong  
 PI WO 2006124627 A2 23 Nov 2006  
 SO (2006) PCT Int. Appl., 49pp.  
 CODEN: PIXXD2  
 CY UNITED STATES  
 DT Patent  
 FS CAPLUS  
 OS CAPLUS 2006:1226364  
 LA English  
 ED Entered STN: 19 Dec 2006  
 Last Updated on STN: 5 Jun 2007

L3 ANSWER 13 OF 13 TOXCENTER COPYRIGHT 2007 ACS on STN  
 AN 2005192720 TOXCENTER  
 CP Copyright 2007 ACS  
 DN CA14306091068P  
 TI Methods of stimulating axonal growth of CNS neurons using Nogo receptor antagonists in combination with growth factors  
 AU Benowitz, Larry I.; Fischer, Dietmar  
 CS ASSIGNEE: Children's Medical Center Corporation  
 PI WO 2005059515 A2 30 Jun 2005  
 SO (2005) PCT Int. Appl., 74 pp.  
 CODEN: PIXXD2  
 CY UNITED STATES  
 DT Patent  
 FS CAPLUS  
 OS CAPLUS 2005:564795  
 LA English  
 ED Entered STN: 19 Jul 2005  
 Last Updated on STN: 9 Jan 2007

=> s reticulon(w)family(w)peptide  
 L4 0 RETICULON(W) FAMILY(W) PEPTIDE  
 L5 459 NOGO(W) RECEPTOR  
 => s 15 (p) (11 or 12)  
 L6 3 L5 (P) (L1 OR L2)  
 => d 16 1-3

L6 ANSWER 1 OF 3 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN  
 AN 2002:602313 BIOSIS  
 DN PREV200200602313  
 TI The neurotrophin receptor p75NTR: Novel functions and implications for

diseases of the nervous system.  
 AU Dechant, Georg; Barde, Yves-Alain [Reprint author]  
 CS Friedrich Miescher Institute for Biomedical Research, Maulbeerstr. 66,  
 4058, Basel, Switzerland  
 yves.barde@fmi.ch  
 SO Nature Neuroscience, (November, 2002) Vol. 5, No. 11, pp. 1131-1136.  
 print.  
 ISSN: 1097-6256.  
 DT Article  
 LA English  
 ED Entered STN: 27 Nov 2002  
 Last Updated on STN: 27 Nov 2002

L6 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2007 ACS on STN  
 AN 2006:152149 CAPLUS  
 DN 144:290750  
 TI Alzheimer precursor protein interaction with the nogo-66 receptor reduces  
 amyloid- $\beta$  plaque deposition  
 AU Park, James H.; Gimbel, David A.; GrandPre, Tadzia; Lee, Jung-Kil; Kim,  
 Ji-Eun; Li, Weiwei; Lee, Daniel H. S.; Strittmatter, Stephen M.  
 CS Department of Neurology, Yale University School of Medicine, New Haven,  
 CT, 06510, USA  
 SO Journal of Neuroscience (2006), 26(5), 1386-1395  
 CODEN: JNRSDS; ISSN: 0270-6474.  
 PB Society for Neuroscience  
 DT Journal  
 LA English  
 RE.CNT 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD  
 ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 3 OF 3 TOXCENTER COPYRIGHT 2007 ACS on STN  
 AN 2002:273799 TOXCENTER  
 CP Copyright (c) 2007 The Thomson Corporation  
 DN PREV20020602313  
 TI The neurotrophin receptor p75NTR: Novel functions and implications for  
 diseases of the nervous system  
 AU Dechant, Georg; Barde, Yves-Alain [Reprint author]  
 CS Friedrich Miescher Institute for Biomedical Research, Maulbeerstr. 66,  
 4058, Basel, Switzerland yves.barde@fmi.ch  
 SO Nature Neuroscience, (November, 2002) Vol. 5, No. 11, pp. 1131-1136.  
 print.  
 ISSN: 1097-6256.  
 DT Article  
 FS BIOSIS  
 OS BIOSIS 2002:602313  
 LA English  
 ED Entered STN: 3 Dec 2002  
 Last Updated on STN: 3 Dec 2002

=> s NGR1(w)antagonist  
 L7 2 NGR1(w) ANTAGONIST  
 => d 17 1-2

L7 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2007 ACS on STN  
 AN 2006:1226364 CAPLUS  
 DN 146:26348  
 TI Neuronal degeneration treatment with Nogo receptor antagonists  
 AU Lee, Daniel H. S.; Sah, Dinah W. Y.; So, Kwok Fai; Wu, Wutian  
 PA Biogen Idec Ma Inc., USA; The University of Hong Kong  
 SO PCT Int. Appl., 49pp.

CODEN: PIXXD2  
 DT Patent  
 LA English  
 FAN.CNT 1  
 PATENT NO. APPLICATION NO. DATE  
 PI WO 2006/124627 A2 20061123 WO 2006-US18484 20060512  
 W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,  
 CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,  
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 PRAI US 2005-679959P 20050512  
 US 2005-735187P P 20051110

L7 ANSWER 2 OF 2 TOXCENTER COPYRIGHT 2007 ACS on STN  
 AN 2006:388140 TOXCENTER  
 CP Copyright 2007 ACS  
 DN CA14602026348C  
 TI Neuronal degeneration treatment with Nogo receptor antagonists  
 AU Lee, Daniel H. S.; Sah, Dinah W. Y.; So, Kwok Fai; Wu, Wutian  
 CS ASSIGNEE: The University of Hong Kong  
 PI WO 2006124627 A2 23 Nov 2006  
 SO (2006) PCT Int. Appl., 49pp.  
 CODEN: PIXXD2.  
 CY UNITED STATES  
 DT Patent  
 FS CAPLUS  
 OS CAPLUS 2006:1226364  
 LA English  
 ED Entered STN: 19 Dec 2006  
 Last Updated on STN: 5 Jun 2007

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 L3 13 S NOGO(W)RECEPTOR(W)ANTAGONIST  
 L4 0 S RETICULON(W)FAMILY(W)PEPTIDE  
 L5 459 S NOGO(W)RECEPTOR  
 L6 3 S L5 (P) (L1 OR L2)  
 L7 2 S NGR1(W)ANTAGONIST  
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 => d 18 1-4

ANSWER 1 OF 4	CAPLUS	COPYRIGHT 2007 ACS on STN
2005:823596	CAPLUS	
143:222540		
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PATENT NO.	KIND	DATE
WO 2005074972	A2	20050818
WO 2005074972	A3	20051222
W:		WO 2005-US2535
AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GE, GH, GM, GR, HU, ID, IL, IN, IS, JP, KE, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MX, MY, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, RW, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AZ, BY, BG, BR, BU, BT, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BU, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, NI, TD, TG		20050128
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EP 1713494	A2	20061025
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CN 1946418	A	20070411
US 2004-540798P	P	20040130
WO 2005-US2535	W	20050128
ANSWER 2 OF 4	CAPLUS	COPYRIGHT 2007 ACS on STN
2005:564795	CAPLUS	
143:91068		
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AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GE, GH, GM, GR, HU, ID, IL, IN, IS, JP, KE, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MX, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, RW, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AZ, BY, BG, BR, BU, BT, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BU, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, NI, TD, TG		20041216
ANSWER 1 OF 4	CAPLUS	COPYRIGHT 2007 ACS on STN
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WO 2005074972	A3	20051222
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AU 2005210621	A1	20050818
CA 2555018	A1	20050818
EP 1713494	A2	20061025
R:		EP 2005-712127
IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, BA, HR, IS, YU		20050128
CN 1946418	A	20070411
US 2004-540798P	P	20040130
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ANSWER 2 OF 4	CAPLUS	COPYRIGHT 2007 ACS on STN
2005:564795	CAPLUS	
143:91068		
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ENT 1		
PATENT NO.	KIND	DATE
WO 2005059515	A2	20050630
WO 2005059515	A3	20060908
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MR. NE, SN, TD, TG	CA 2549000	A1	20050630	CA 2004-2549000	20041216
EP 1695061	A2	20060830	EP 2004-814439	20041216	20041216
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, BA, HR, IS, YU					
JP 2007514748	T	20070607	JP 2006-545428	20041216	20041216
PPRAI US 2003-529833P	P	20031216			
WO 2004-US42255	W	20041216			
ANSWER 3 OF 4 CAPLUS COPYRIGHT 2007 ACS on STN					
LAN 2004:927061 CAPLUS					
AN 141:406109					
DTI Treatment of conditions involving amyloid plaques					
TTI Strittmatter, Stephen M.; Lee, Daniel H. S.; Li, Weiwei					
IN USA					
SO PCT Int. Appl., 43 pp.					
PA CODEN: PIXXD2					
DT Patent					
LA English					
AFAN CNT 1					
PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
WO 2004093893	A2	20041104	WO 2004-US11728	20040416	
WO 2004093893	A3	20050303			
W: AE, AL, AG, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW, BW, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GN, GQ, GW, ML, MR, NE, SN, TD, TG					
AU 2004231742	A2	20041104	AU 2004-231742	20040416	
AU 2004231742	A1	20041104			
CA 2522649	A1	20041104	CA 2004-2522649	20040416	
EP 1615654	A2	20060118	EP 2004-759905	20040416	
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR					
BR 2004003562	A	20060418	BR 2004-9562	20040416	
CN 1832752	A	20060913	CN 2004-80016919	20040416	
JP 2006523708	T	20061019	JP 2006-510107	20040416	
NO 2005005392	A	20051115	NO 2005-5392	20051115	
US 2007065429	A1	20070332	US 2006-553669	20060809	
US 2003-463424P	P	20030416			
PPRAI WO 2004-US11728	W	20040416			
ANSWER 4 OF 4 CAPLUS COPYRIGHT 2007 ACS on STN					
LAN 2004:142908 CAPLUS					
AN 140:198086					
DTI Nogo receptor antagonists for promoting					
TTI survival of neuron and treating multiple sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury					
IN Lee, Daniel H. S.; Pepinsky, R. Blake; Li, Weiwei; Rabacchi, Sylvia A.; Relton, Jane K.; Worley, Dane S.; Strittmatter, Stephen M.; Sah, Dinah Y. W.					
PA Yale University, USA; Biogen, Inc.					
SO PCT Int. Appl., 133 pp.					
LA CODEN: PIXXD2					
DT Patent					
LA English					

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=> s l10 and alzheimer
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L13 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2007 ACS on STN  
 PAN 2006:113021 CAPLUS  
 DDN 144:101055  
 TTI Protein Sp35/*LINGO-1* antagonists for  
 treatment of conditions involving demyelination  
 IN Mi. Sha; Pepinsky, R. Blake; McCoy, John  
 PA Biogen Idec Ma Inc., USA  
 SO PCT Int. Appl., 183 pp.  
 CODEN: PIXXD2  
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AB . . . the thalamus in RHRSP was observed at 1, 2 and 4 weeks after  
AB . . . distal MCAO. In addition, intracerebroventricular infusion of

the expression of Nogo-A in oligodendrocytes increased persistently and its localization became redistributed around damaged axons and dendrites.

the expression of Nogo-A in oligodendrocytes increased persistently and its localization became redistributed around damaged axons and dendrites. Administration of *NEP1-40* downregulated the expression

**Administration of NEP1-40 downregulated the expression of Nogo-A, reduced axonal injury and enhanced axonal regeneration. Our data suggest that Nogo-A is involved in . . .**

of Nogo-A, reduced axonal injury and enhanced axonal regeneration. Our data suggest that Nogo-A is involved in . . .

- recovery.
- L14 ANSWER 2 OF 25 MEDLINE on STN  
AB . . . inhibitors (MAI). To overcome CSPG- or myelin-induced inhibition, strategies based on extrinsic and intrinsic treatments have been developed. For example, *NEP1-40* is a synthetic peptide that promotes axonal regeneration by blocking Nogo-66/Ngr interaction and chondroitinase ABC (ChABC), which degrades CS, thereby, model, overexpressed CSPG and MAI impaired axon regrowth, which mimics regeneration failure in vivo. Both CS cleavage with ChABC and *NEP1-40* strongly facilitated the regrowth of entorhinal axons after axotomy permitting the re-establishment of synaptic contacts with target cells. However, the combined treatment did not improve the axonal regrowth compared with acute treatment. These results provide insight into the development of .
- CN 0 (Myelin Proteins); 0 (*NEP1-40* protein, human); 0 (Nogo protein); 0 (Peptide Fragments); 0 (Proteochondroitin Sulfates); 0 (Receptors, Cell Surface); 0 (Rtnr protein, mouse); EC.
- L14 ANSWER 3 OF 25 MEDLINE on STN  
TI Cloning of *NEP1-40* gene and expression of its protein.  
AB OBJECTIVE: To clone the genes of nogo-66 and *NEP1-40* from spinal cord of rat and to realize the expression of its protein in vitro. METHODS: The nogo-66 and *NEP1-40* genes were cloned from the spinal cord of juvenile rat by use of RT-PCR techniques, and the objective genes were . . . express the proteins. The two proteins were purified by Ni-column and detected by using Western-blot test. RESULTS: The Nogo-66 and *NEP1-40* genes were successfully cloned from rat, which were 215 bp and 137 bp for each one when add the enzyme. . . the results of electrophoresis. The expression plasmids were induced by IPTG and got the purified GST fusion protein nogo-66 and *NEP1-40*, which relative molecular weight were 33.2 x 10(3) and 30.3 x 10(3) respectively. The results of Western-blot test confirmed that the antigenicity of the two proteins was precise. CONCLUSION: Nogo-66 and *NEP1-40* proteins can be expressed in a high efficiency in vitro using genetic engineering, so it provides a good basis for .
- L14 ANSWER 4 OF 25 MEDLINE on STN  
AB IN-1, the monoclonal antibody against the exon 3-encoded N-terminal domain of Nogo-A, and the Nogo-66 receptor (NGR) antagonist *NEP1-40* have both shown efficacy in promoting regeneration in animal spinal cord injury models, the latter even when administered subcutaneously 1. . . targeted disruption of Nogo and Ngr have, surprisingly, only modest regenerative capacity (if any) compared with treatment with IN-1 or *NEP1-40*. Disruption of the Nogo gene by various groups yielded results ranging from significant regenerative improvement in young mice to number . . . background, we suggest here some possible and testable explanations for the above phenomena. These possibilities include effects of IN-1 and *NEP1-40* on the CNS beyond neutralization of Nogo and Ngr functions, and the latter's possible role in the CNS beyond that.
- L14 ANSWER 5 OF 25 MEDLINE on STN  
AB . . . approach can be adapted to systemic therapy in a postinjury therapeutic time window. Subcutaneous treatment with the Ngr antagonist peptide *NEP1-40* (Nogo extracellular peptide, residues 1-40) results in extensive growth of corticospinal axons, sprouting of serotonergic fibers, upregulation of axonal growth. . . protein 1A), and synapse re-formation. Locomotor recovery after thoracic spinal cord injury is enhanced. Furthermore, delaying the initiation of systemic *NEP1-40* administration for up to 1 week after cord lesions does not limit the degree of axon sprouting and functional
- L14 ANSWER 6 OF 25 MEDLINE on STN  
AB . . . Ngr. Here, we identify competitive antagonists of Ngr derived from amino-terminal peptide fragments of Nogo-66. The Nogo-66(1-40) antagonist peptide (*NEP1-40*) blocks Nogo-66 or CNS myelin inhibition of axonal outgrowth in vitro, demonstrating that Ngr mediates a significant portion of axonal outgrowth inhibition by myelin. Intrathecal administration of *NEP1-40* to rats with mid-thoracic spinal cord hemisection results in significant axon growth of the corticospinal tract, and improves functional recovery. Thus, Nogo-66 and Ngr have central roles in limiting axonal regeneration after CNS injury, and *NEP1-40* provides a potential therapeutic agent.
- L14 ANSWER 7 OF 25 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN  
AB. . . inhibitors (MAI). To overcome CSPG- or myelin-induced inhibition, strategies based on extrinsic and intrinsic treatments have been developed. For example, *NEP1-40* is a synthetic peptide that promotes axonal regeneration by blocking Nogo-66/Ngr interaction and chondroitinase ABC (ChABC), which degrades CS, thereby, model, overexpressed CSPG and MAI impaired axon regrowth, which mimics regeneration failure in vivo. Both CS cleavage with ChABC and *NEP1-40* strongly facilitated the regrowth of entorhinal axons after axotomy, permitting the re-establishment of synaptic contacts with target cells. However, the combined treatment did not improve the regeneration induced by ChABC alone, and the delayed treatment of ChABC, but not *NEP1-40*, had a less pronounced effect on axonal regrowth compared with acute treatment. These results provide insight into the development of .
- IT nervous system  
IT Chemicals & Biochemicals  
chondroitin sulfate proteoglycans; chondroitinase ABC [EC 4.2.2.4]; myelin-associated glycoprotein; Nogo-66 receptor; oligodendrocyte myelin glycoprotein; *NEP1-40*
- L14 ANSWER 8 OF 25 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN  
AB IN-1, the monoclonal antibody against the exon 3-encoded N-terminal domain of Nogo-A, and the Nogo-66 receptor (NGR) antagonist *NEP1-40* have both shown efficacy in promoting regeneration in animal spinal cord injury models, the latter even when administered subcutaneously 1. . . targeted disruption of Nogo and Ngr have, surprisingly, only modest regenerative capacity (if any) compared with treatment with IN-1 or *NEP1-40*. Disruption of the Nogo gene by various groups yielded results ranging from significant regenerative improvement in young mice to number . . . background, we suggest here some possible and testable explanations for the above phenomena. These possibilities include effects of IN-1 and *NEP1-40* on the CNS beyond neutralization of Nogo and Ngr functions, and the latter's possible role in the CNS beyond that.
- IT system disease, injury  
IT Spinal Cord Injuries (MeSH)  
Chemicals & Biochemicals  
Nogo receptor; Nogo-66 receptor; neutralizing agent; IN-1; Nogo-A; N-terminal domain; *NEP1-40*; immunologic-drug, immunostimulant-drug, subcutaneous administration
- L14 ANSWER 9 OF 25 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN  
AB. . . of CS myelin than those that target specific myelin proteins e.g. the anti-NogoA antibody, IN-1 or the NogoA derived peptide, *NEP1-40*, even though both reagents successfully promote neurite outgrowth or axonal regeneration in vitro and in vivo. Recently, two Ngr1

- homologues.
- IT CNS: nervous system; DRG neurites; DRG neurons: nervous system; neurites: nervous system
- IT Chemicals & Biochemicals  
CNS myelin; IN-1; MAG; **NEP1-40**; Nogo; Nogo66;  
NogoA; antibodies; leucine; myelin protein; p75; rhoA
- L14 ANSWER 10 OF 25 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on  
STN  
AB. competitive binding between AP-Nogo66 and MAG in CHO cells overexpressing NGR1, whereas Liu et al. (2002, Science 297:1190-3) showed that **NEP1-40** (a peptide derived from Nogo66) was unable to block MAG binding to NGR1 but successfully blocked AP-Nogo66. We compared MAG.
- IT Organisms  
CNS: nervous system; membrane; neurons: nervous system;  
Oligodendrocytes: nervous system
- IT Chemicals & Biochemicals  
GPI; GST; MAG [myelin-associated glycoprotein]; **NEP1-40**; Nogo-A; Nogo66; SPA; glycoprotein; membrane protein; myelin protein
- L14 ANSWER 11 OF 25 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on  
STN  
AB. conclusion about the role of Nogo-66 or its receptor (NGR). We identify a peptide antagonist (Nogo Extracellular Peptide residues 1-40, **NEP1-40**) of the Nogo-66 Receptor derived from amino terminal fragments of the Nogo-66 domain. This antagonist binds to the Nogo-66 Receptor. These findings reveal the central role of the Nogo-66 Receptor in limiting axonal regeneration after adult mammalian CNS injury, and **NEP1-40** provides a potential therapeutic approach to treating traumatic CNS axonal injury.
- IT nervous system
- IT Diseases  
Spinal cord injury: injury, nervous system disease, drug therapy  
Spinal Cord Injuries (MeSH)
- IT Chemicals & Biochemicals  
**NEP1-40** [Nogo extracellular peptide residues 1-40]:  
central stimulant-drug, neuroprotectant-drug, pharmacodynamics; Nogo-66 receptor: therapeutic recovery; myelin
- L14 ANSWER 12 OF 25 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on  
STN  
AB. approach can be adapted to systemic therapy in a postinjury therapeutic time window. Subcutaneous treatment with the NGR antagonist peptide **NEP1-40** (Nogo extracellular peptide, residues 1-40) results in extensive growth of corticospinal axons, sprouting of serotonergic fibers, upregulation of axonal growth. protein 1A), and synapse re-formation. Locomotor recovery after thoracic spinal cord injury is enhanced. Furthermore, delaying the initiation of systemic **NEP1-40** administration for up to 1 week after cord lesions does not limit the degree of axon sprouting and functional recovery.
- IT serotonergic fiber: nervous system
- IT Diseases  
Spinal cord injury: injury, nervous system disease  
Spinal Cord Injuries (MeSH)
- IT Chemicals & Biochemicals  
**NEP1-40** peptide; Nogo; Nogo-66 receptor antagonist;  
delayed systemic administration; SPRLA [small proline-rich repeat
- protein 1A]
- L14 ANSWER 13 OF 25 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on  
STN  
AB. or NGR. Here, we identify competitive antagonists of NGR derived from amino-terminal peptide fragments of Nogo-66. The Nogo-66(1-40) antagonist peptide (**NEP1-40**) blocks Nogo-66 or CNS myelin inhibition of axonal outgrowth in vitro, demonstrating that NGR mediates a significant portion of axonal outgrowth inhibition by myelin. Intrathecal administration of **NEP1-40** to rats with mid-thoracic spinal cord hemisection results in significant axon growth of the corticospinal tract, and improves functional recovery. Thus, Nogo-66 and NGR have central roles in limiting axonal regeneration after CNS injury, and **NEP1-40** provides a potential therapeutic agent.
- L14 ANSWER 14 OF 25 CAPLUS COPYRIGHT 2007 ACS on STN  
AB. the thalamus in RHSP was observed at 1, 2 and 4 wk after distal MCAO. In addition, intracerebroventricular infusion of **NEP1-40**, a Nogo-66 receptor (NGR) antagonist peptide, was administered starting 24 h after MCAO and continued for 1, 2 and 4. the expression of Nogo-A in oligodendrocytes increased persistently and its localization became redistributed around damaged axons and dendrites. Administration of **NEP1-40** downregulated the expression of Nogo-A, reduced axonal injury and enhanced axonal regeneration. Our data suggest that Nogo-A is involved in.
- IT Injury  
axon; **NEP1-40** reduced axonal injury and enhanced axonal regeneration)
- IT Axon  
(disease, injury; **NEP1-40** reduced axonal injury and enhanced axonal regeneration)
- IT Axon  
(regeneration; **NEP1-40** reduced axonal injury and enhanced axonal regeneration)
- L14 ANSWER 15 OF 25 CAPLUS COPYRIGHT 2007 ACS on STN  
TI Cloning of **NEP1-40** gene and expression of its protein  
AB The genes of nogo-66 and **NEP1-40** from spinal cord of rat were cloned and the expression of its protein in vitro was studied. The nogo-66 and **NEP1-40** genes were cloned from the spinal cord of juvenile rat by RT-PCR techniques, and the objective genes were bonded to . proteins. The two proteins were purified by Ni-column and detected by using Western-blot test. Results showed that the Nogo-66 and **NEP1-40** genes were successfully cloned from rat, which were 215 bp and 137 bp for each one when add the enzyme. the results of electrophoresis. The expression plasmids were induced by IPTG and got the purified GST fusion protein nogo-66 and **NEP1-40**, which relative mol. weight were 33.2+103 and 30.3+103, resp. The results of Western-blot test confirmed that the antigenicity of the two proteins was precise. It was conclusion that Nogo-66 and **NEP1-40** proteins could be expressed in high efficiency in vitro using genetic engineering, which provided a good basis for further research.
- L14 ANSWER 16 OF 25 CAPLUS COPYRIGHT 2007 ACS on STN  
AB. glycoprotein) and OMgp (oligodendrocyte-myelin glycoprotein) in spinal cord injury (SCI) and the immunotherapy for SCI with monoclonal antibodies of Nogo-A, **NEP1-40** and DNA vaccines, etc.
- L14 ANSWER 17 OF 25 CAPLUS COPYRIGHT 2007 ACS on STN  
AB. inhibitors (MAI). To overcome CSPG- or myelin-induced inhibition, strategies based on extrinsic and intrinsic treatments have been developed. For example, **NEP1-40** is a synthetic



peptide that promotes axonal regeneration by blocking Nogo-66/NGR interaction and chondroitinase ABC (ChABC), which degrades CS, thereby, model, overexpressed CSPG and MAI impaired axon regrowth, which mimics regeneration failure in vivo. Both CS cleavage with ChABC and NRP1-40 strongly facilitated the regrowth of entorhinal axons after axotomy, permitting the re-establishment of synaptic contacts with target cells. However, the combined treatment did not improve the regeneration induced by ChABC alone, and the delayed treatment of ChABC, but not NRP1-40, had a less pronounced effect on axonal regrowth compared with acute treatment. These results provide insight into the development of regeneration entorhino hippocampal axon degrading proteoglycan signaling; chondroitinase ABC NRP1-40 entorhino hippocampal axon regeneration

ST

L14 ANSWER 18 OF 25 CAPLUS COPYRIGHT 2007 ACS on STN  
AB A review. IN-1, the monoclonal antibody against the exon 3-encoded N-terminal domain of Nogo-A, and the Nogo-66 receptor (NGR) antagonist NRP1-40 have both shown efficacy in promoting regeneration in animal spinal cord injury models, the latter even when administered s.c. 1. . . targeted disruption of Nogo and NGR have, surprisingly, only modest regenerative capacity (if any) compared with treatment with IN-1 or NRP1-40. Disruption of the Nogo gene by various groups yielded results ranging from significant regenerative improvement in young mice to number . . . background, we suggest here some possible and testable explanations for the above phenomena. These possibilities include effects of IN-1 and NRP1-40 on the CNS beyond neutralization of Nogo and NGR functions, and the latter's possible role in the CNS beyond that.

L14 ANSWER 19 OF 25 CAPLUS COPYRIGHT 2007 ACS on STN  
AB . . . approach can be adapted to systemic therapy in a postinjury therapeutic time window. S.C. treatment with the NGR antagonist peptide NRP1-40 (Nogo extracellular peptide, residues 1-40) results in extensive growth of corticospinal axons, sprouting of serotonergic fibers, upregulation of axonal growth, sprouting of and synapse re-formation. Locomotor recovery after thoracic spinal cord injury is enhanced. Furthermore, delaying the initiation of systemic NRP1-40 administration for up to 1 wk after cord lesions does not limit the degree of axon sprouting and functional recovery.

L14 ANSWER 20 OF 25 CAPLUS COPYRIGHT 2007 ACS on STN  
AB . . . or NGR. Here, we identify competitive antagonists of NGR derived from amino-terminal peptide fragments of Nogo-66. The Nogo-66(1-40) antagonist peptide (NRP1-40) blocks Nogo-66 or CNS myelin inhibition of axonal outgrowth in vitro, demonstrating that NGR mediates a significant portion of axonal outgrowth inhibition by myelin. Intrathecal administration of NRP1-40 to rats with mid-thoracic spinal cord hemisection results in significant axon growth of the corticospinal tract, and improves functional recovery. Thus, Nogo-66 and NGR have central roles in limiting axonal regeneration after CNS injury, and NRP1-40 provides a potential therapeutic agent.

L14 ANSWER 21 OF 25 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN  
AB . . . the thalamus in RHSP was observed at 1, 2 and 4 weeks after distal MCAO. In addition, intracerebroventricular infusion of NRP1-40, a Nogo-66 receptor (NGR) antagonist peptide, was administered starting 24 h after MCAO and continued for 1, 2 and 4. . . the expression of Nogo-A in oligodendrocytes increased persistently and its localization became redistributed around damaged axons and dendrites. Administration of NRP1-40 downregulated the expression

of Nogo-A, reduced axonal injury and enhanced axonal regeneration. Our data suggest that Nogo-A is involved in.

CT

Medical Descriptors:  
animal . . . localization  
rat  
stroke prone spontaneously hypertensive rat  
thalamus ventral nucleus  
Nogo 66 receptor  
amyloid precursor protein: EC, endogenous compound  
microtubule associated protein 2: EC, endogenous compound  
nep1 40: DV, drug development  
nep1 40: DT, drug therapy  
nep1 40: CV, intracerebroventricular drug administration  
nep1 40: PD, pharmacology  
neuromodulin: EC, endogenous compound  
\*protein Nogo A: EC, endogenous compound  
receptor blocking agent: DV, drug development  
receptor blocking agent: DT, drug.  
(1) Nep1 40

CN

L14 ANSWER 22 OF 25 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN  
AB IN-1, the monoclonal antibody against the exon 3-encoded N-terminal domain of Nogo-A, and the Nogo-66 receptor (NGR) antagonist NRP1-40 have both shown efficacy in promoting regeneration in animal spinal cord injury models, the latter even when administered subcutaneously 1. . . targeted disruption of Nogo and NGR have, surprisingly, only modest regenerative capacity (if any) compared with treatment with IN-1 or NRP1-40. Disruption of the Nogo gene by various groups yielded results ranging from significant regenerative improvement in young mice to number . . . background, we suggest here some possible and testable explanations for the above phenomena. These possibilities include effects of IN-1 and NRP1-40 on the CNS beyond neutralization of Nogo and NGR functions, and the latter's possible role in the CNS beyond that.

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CT Medical Descriptors:

\*central . . . comparison  
monoclonal antibody 7B12: DV, drug development  
monoclonal antibody 7B12: DT, drug therapy  
monoclonal antibody 7B12: PD, pharmacology  
monoclonal antibody 7B12: IV, intravenous drug administration  
monoclonal antibody NRP1 40: CM, drug comparison  
monoclonal antibody NRP1 40: DV, drug development  
monoclonal antibody NRP1 40: DT, drug therapy  
monoclonal antibody NRP1 40: PD, pharmacology  
monoclonal antibody NRP1 40: IP, intraperitoneal drug administration  
monoclonal antibody NRP1 40: SC, subcutaneous drug administration  
monoclonal antibody AS472: CM, drug comparison  
monoclonal antibody AS472: DV, drug development  
monoclonal antibody AS472: DT.

L14 ANSWER 24 OF 25 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

AB . . . approach can be adapted to systemic therapy in a postinjury therapeutic time window. Subcutaneous treatment with the NGR antagonist peptide NRP1-40 (Nogo extracellular peptide, residues 1-40) results in extensive growth of corticospinal axons, sprouting of serotonergic fibers, upregulation of axonal growth, . . . protein 1A), and synapse re-formation. Locomotor recovery after thoracic spinal cord

injury is enhanced. Furthermore, delaying the initiation of systemic NPEP1-40 administration for up to 1 week after cord lesions does not limit the degree of axon sprouting and functional recovery.

L14 ANSWER 25 OF 25 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN  
AB . . . or Ngr. Here, we identify competitive antagonists of Ngr derived from aminoterminal peptide fragments of Nogo-66. The Nogo-66(1-40) antagonist peptide (NPEP1-40) blocks Nogo-66 or CNS myelin inhibition of axonal outgrowth in vitro, demonstrating that Ngr mediates a significant portion of axonal outgrowth inhibition by myelin. Intrathecal administration of NPEP1-40 to rats with thoracic spinal cord hemisection results in significant axon growth of the corticospinal tract, and improves functional recovery. Thus, Nogo-66 and Ngr have central roles in limiting axonal regeneration after CNS injury, and NPEP1-40 provides a potential therapeutic agent.

=> s l14 and (l1 or l2)  
L15 0 L14 AND (L1 OR L2)  
=> s l14 and alzheimer  
L16 0 L14 AND ALZHEIMER  
=> d l14 1-25

L14 ANSWER 1 OF 25 MEDLINE on STN  
AN 2007239440 IN-PROCESS  
DN PubMed ID: 17382469  
TI Nogo-A is involved in secondary axonal degeneration of thalamus in hypertensive rats with focal cortical infarction.  
AU Wang Fang; Liang Zhijian; Hou Qinghua; Xing Shihui; Ling Li; He Meixia; Pei Zhong; Zeng Jinsheng  
CS Department of Neurology and Stroke Center, The First Affiliated Hospital, Sun Yat-Sen University, No. 58 Zhongshan Road 2, Guangzhou 510080, China.  
SO Neuroscience Letters. (2007 May 7) Vol. 417, No. 3, pp. 255-60.  
Electronic Publication: 2007-03-12.  
Journal code: 7600130. ISSN: 0304-3940.

CY Ireland  
DT Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, NON-U.S. GOV'T)  
LA English  
FS NONMEDLINE; IN-PROCESS; NONINDEXED; Priority Journals  
ED Entered STN: 24 Apr 2007  
Last Updated on STN: 13 Jun 2007

L14 ANSWER 2 OF 25 MEDLINE on STN  
AN 2006120738 MEDLINE  
DN PubMed ID: 16407455  
TI Regeneration of lesioned entorhino-hippocampal axons in vitro by combined degradation of inhibitory proteoglycans and blockade of Nogo-66/Ngr signaling.  
AU Mingorance Ana; Sole Marta; Muneton Vilma; Martinez Albert; Nieto-Sampedro Manuel; Soriano Eduardo; del Rio Jose Antonio  
CS Development and Regeneration of the CNS, Department of Cell Biology, IIR-PCB, University of Barcelona, Barcelona, Spain.  
SO The FASEB Journal : official publication of the Federation of American Societies for Experimental Biology. (2006 Mar) Vol. 20, No. 3, pp. 491-3.  
Electronic Publication: 2006-01-11.  
Journal code: 8804484. E-ISSN: 1530-6860.  
CY United States  
DT (IN VITRO)

Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, NON-U.S. GOV'T)  
LA English  
FS Priority Journals  
EM 200604  
ED Entered STN: 2 Mar 2006  
Last Updated on STN: 22 Apr 2006  
Entered Medline: 21 Apr 2006

L14 ANSWER 3 OF 25 MEDLINE on STN  
AN 2006071862 IN-PROCESS  
DN PubMed ID: 16457436  
TI Cloning of NPEP1-40 gene and expression of its protein.  
AU Gong Fullang; Wang Kunzheng; Yu Fengbo  
CS Department of Orthopaedic Surgery, Second Hospital of Xi'an Jiaotong University, Xi'an Shaanxi, 710004, PR China.  
SO Zhongguo xiu fu chong jian wai ke za zhi = Zhongguo xiu fu chong jian wai ke zhi = Chinese journal of reparative and reconstructive surgery. (2006 Jan) Vol. 20, No. 1, pp. 9-12.  
Journal code: 9425194. ISSN: 1002-1892.

CY China  
DT (ENGLISH ABSTRACT)  
Journal; Article; (JOURNAL ARTICLE)  
LA Chinese  
FS NONMEDLINE; IN-PROCESS; NONINDEXED; Priority Journals  
ED Entered STN: 7 Feb 2006  
Last Updated on STN: 12 Dec 2006

L14 ANSWER 4 OF 25 MEDLINE on STN  
AN 2005427718 MEDLINE  
DN PubMed ID: 16092935  
TI Why do Nogo/Nogo-66 receptor gene knockouts result in inferior regeneration compared to treatment with neutralizing agents? .  
AU Teng Feilicia Yu Hsuan; Tang Bor Luen  
CS Department of Biochemistry and Programme in Neurobiology and Aging, National University of Singapore, Singapore.  
SO Journal of neurochemistry. (2005 Aug) Vol. 94, No. 4, pp. 865-74. Ref: 70  
Journal code: 2985190R. ISSN: 0022-3042.

CY England; United Kingdom  
DT (COMPARATIVE STUDY)  
Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, NON-U.S. GOV'T)  
General Review; (REVIEW)

LA English  
FS Priority Journals  
EM 200509  
ED Entered STN: 15 Aug 2005  
Last Updated on STN: 28 Sep 2005  
Entered Medline: 27 Sep 2005

L14 ANSWER 5 OF 25 MEDLINE on STN  
AN 2003241538 MEDLINE  
DN PubMed ID: 12764110  
TI Delayed systemic Nogo-66 receptor antagonist promotes recovery from spinal cord injury.  
AU Li Shuxin; Strittmatter Stephen M  
CS Department of Neurology and Section of Neurobiology, Yale University School of Medicine, New Haven, Connecticut 06520, USA.  
SO The Journal of neuroscience : the official journal of the Society for Neuroscience. (2003 May 15) Vol. 23, No. 10, pp. 4219-27.  
Journal code: 8102140. E-ISSN: 1529-2401.  
CY United States  
DT Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, NON-U.S. GOV'T)

(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)

LA English

FS Priority Journals

EM 200306

ED Entered STN: 24 May 2003

Last Updated on STN: 26 Jun 2003

Entered Medline: 25 Jun 2003

L14 ANSWER 6 OF 25 MEDLINE ON STN

AN 2002297070 MEDLINE

DN PubMed ID: 12037567

TI Nogo-66 receptor antagonist peptide promotes axonal regeneration.

AU GrandPre Tadiia, Li Shuxin, Strittmatter Stephen M

CS Department of Neurology and Section of Neurobiology, Yale University

SO School of Medicine, New Haven, CT 06520, USA.

Nature, (2002 May 30) Vol. 417, No. 6888, pp. 547-51.

Journal code: 0410462. ISSN: 0028-0836.

CY England: United Kingdom

Journal, Article: (JOURNAL ARTICLE)

DT (RESEARCH SUPPORT, NON-U.S. GOV'T)

(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)

LA English

FS Priority Journals

EM 200206

ED Entered STN: 31 May 2002

Last Updated on STN: 28 Jun 2002

Entered Medline: 27 Jun 2002

L14 ANSWER 7 OF 25 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

AN 2006:305359 BIOSIS

DN PREV200600300324

TI Regeneration of lesioned entorhino-hippocampal axons in vitro by combined

degradation of inhibitory proteoglycans and blockade of Nogo-66/NGR

signaling.

AU Mingorance, Ana, Sole, Marta, Muneton, Vilma; Martinez, Albert;

Nieto-Sampedro, Manuel; Soriano, Eduardo; del Rio, Jose Antonio [Reprint

Author]

CS Univ Barcelona, IIR PCB, Dept Cell Biol, Dev and Regenerat CNS, Barcelona

Sci Pk, Josep Samitier 1-5, E-08028 Barcelona, Spain

jario@pcb.uib.es

SO FASEB Journal, (JAN 2006) Vol. 20, No. 1.

CODEN: FAJOC. ISSN: 0892-6638.

DT Article

LA English

ED Entered STN: 7 Jun 2006

Last Updated on STN: 7 Jun 2006

L14 ANSWER 8 OF 25 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

AN 2005:440461 BIOSIS

DN PREV20051022444

TI Why do Nogo/Nogo-66 receptor gene knockouts result in inferior

regeneration compared to treatment with neutralizing agents?

AU Huan, Felicia Yu; Tang, Bor Luen [Reprint Author]

CS Natl Univ Singapore, Dept Biochem, 8 Med Dr, Singapore 117597, Singapore

behtblenus.edu.sg

SO Journal of Neurochemistry. (AUG 2005) Vol. 94, No. 4, pp. 865-874.

CODEN: JONRA9. ISSN: 0022-3042.

DT Article

LA English

ED Entered STN: 26 Oct 2005

Last Updated on STN: 26 Oct 2005

L14 ANSWER 9 OF 25 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

2004:203037 BIOSIS

DN PREV200400203580

TI Neutralization of Ngr1 may be sufficient to promote rat DRG neurite

outgrowth in the presence of CNS myelination.

AU Li, W. [Reprint Author]; Rabacchi, S. [Reprint Author]; Liu, B.; Pepinsky,

B. [Reprint Author]; Jirik, A. [Reprint Author]; Choi, E. [Reprint

Author]; Morley, D. [Reprint Author]; Friedman, J. [Reprint Author];

Mullen, C. [Reprint Author]; Walus, L. [Reprint Author]; Benedetti, N.

[Reprint Author]; Shao, Z. [Reprint Author]; Levesque, M. [Reprint

Author]; Mi, S. [Reprint Author]; Cate, R. [Reprint Author]; Sah, D.

[Reprint Author]; Strittmatter, S.; Lee, D. [Reprint Author]

CS Dept. Neurodegeneration, Biogen Inc. Cambridge, MA, USA

SO Society for Neuroscience Abstract Viewer and Itinerary Planner, (2003)

Vol. 2003, pp. Abstract No. 678.3. <http://sfn.scholarone.com>. e-file.

Meeting Info.: 33rd Annual Meeting of the Society of Neuroscience. New

Orleans, LA, USA. November 08-12, 2003. Society of Neuroscience.

DT Conference; (Meeting)

LA English

ED Entered STN: 14 Apr 2004

Last Updated on STN: 14 Apr 2004

L14 ANSWER 10 OF 25 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on

STN

AN 2004:203036 BIOSIS

DN PREV200400203579

TI Do MAG and Nogo66 compete for binding to Ngr1?

AU Jirik, A. P. [Reprint Author]; Li, W. [Reprint Author]; Pepinsky, B.

[Reprint Author]; Walus, L. [Reprint Author]; Wang, X. [Reprint Author];

Yang, W. [Reprint Author]; Sah, D. W. Y. [Reprint Author]; Lee, D. H. S.

[Reprint Author]; Rabacchi, S. A. [Reprint Author]

CS Neurobiol., Cambridge, MA, USA

SO Society for Neuroscience Abstract Viewer and Itinerary Planner, (2003)

Vol. 2003, pp. Abstract No. 678.2. <http://sfn.scholarone.com>. e-file.

Meeting Info.: 33rd Annual Meeting of the Society of Neuroscience. New

Orleans, LA, USA. November 08-12, 2003. Society of Neuroscience.

DT Conference; (Meeting)

LA English

ED Entered STN: 14 Apr 2004

Last Updated on STN: 14 Apr 2004

L14 ANSWER 11 OF 25 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on

STN

AN 2003:303647 BIOSIS

DN PREV200300303647

TI Nogo - 66 RECEPTOR ANTAGONIST PEPTIDE PROMOTES AXONAL REGENERATION AND

FUNCTIONAL RECOVERY AFTER SPINAL CORD INJURY.

AU Li, S. [Reprint Author]; GrandPre, T. [Reprint Author]; Strittmatter, S.

M. [Reprint Author]

CS Dept of Neurology, Yale University, New Haven, CT, USA

SO Society for Neuroscience Abstract Viewer and Itinerary Planner, (2002)

Vol. 2002, pp. Abstract No. 203.4. <http://sfn.scholarone.com>. cd-rom.

Meeting Info.: 32nd Annual Meeting of the Society for Neuroscience.

Orlando, Florida, USA. November 02-07, 2002. Society for Neuroscience.

DT Conference; (Meeting)

LA English

ED Entered STN: 2 Jul 2003

Last Updated on STN: 2 Jul 2003

L14 ANSWER 12 OF 25 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on

STN

AN 2003:281722 BIOSIS

DN PREV200300281722  
 TI Delayed systemic Nogo-66 receptor antagonist promotes recovery from spinal cord injury  
 AU Li, Shuxin; Strittmatter, Stephen M. [Reprint Author]  
 CS Department of Neurology and Section of Neurobiology, Yale University School of Medicine, P.O. Box 208018, New Haven, CT, 06520, USA  
 SO Stephen.strittmatter@yale.edu  
 DN ISSN: 0270-6474 (ISSN print)  
 TI Article  
 LA English  
 ED Entered STN: 19 Jun 2003  
 Last Updated on STN: 19 Jun 2003

L14 ANSWER 13 OF 25 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN  
 AN 2002:430039 BIOSIS  
 DN PREV200200430039  
 TI Nogo-66 receptor antagonist peptide promotes axonal regeneration.  
 AU GrandPre, Tadzia; Li, Shuxin; Strittmatter, Stephen M. [Reprint author]  
 CS Department of Neurology and Section of Neurobiology, Yale University School of Medicine, P.O. Box 208018, New Haven, CT, 06520, USA  
 SO Stephen.strittmatter@yale.edu  
 DN ISSN: 0270-6474 (ISSN print)  
 TI Article  
 LA English  
 ED Entered STN: 14 Aug 2002  
 Last Updated on STN: 14 Aug 2002

L14 ANSWER 14 OF 25 CAPLUS COPYRIGHT 2007 ACS on STN  
 AN 2007:448022 CAPLUS  
 TI Nogo-A is involved in secondary axonal degeneration of thalamus in hypertensive rats with focal cortical infarction  
 AU Hong, Fang; Liang, Zhiqian; Hou, Qinghua; Xing, Shihui; Ling, Li; He, Meixia; Pei, Zhong; Zeng, Jinsheng  
 CS Department of Neurology and Stroke Center, The First Affiliated Hospital, Sun Yat-Sen University, Guangzhou, 510080, Peop. Rep. China  
 SO Neuroscience Letters (2007), 417(3), 255-260  
 DN CODEN: NELED5; ISSN: 0304-3940  
 TI Article  
 LA English  
 ED Entered STN: 14 Aug 2002  
 Last Updated on STN: 14 Aug 2002

L14 ANSWER 15 OF 25 CAPLUS COPYRIGHT 2007 ACS on STN  
 AN 2007:263346 CAPLUS  
 TI Cloning of NEFI-40 gene and expression of its protein  
 AU Gong, Fuliang; Wang, Kunzheng; Yu, Pengbo; Dang, Xiaodan; Wang, Chunsheng; Shi, Zhibin; Yang, Pei  
 CS The Second Hospital, Xian Jiaotong University, Xian, Shanxi Province, 710004, Peop. Rep. China  
 SO Zhongguo Xifeng Chongjian Waikexi (2006), 20(1), 9-12  
 DN CODEN: ZXCZEH; ISSN: 1002-1892  
 TI Article  
 LA English  
 ED Entered STN: 14 Aug 2002  
 Last Updated on STN: 14 Aug 2002

L14 ANSWER 16 OF 25 CAPLUS COPYRIGHT 2007 ACS on STN  
 AN 2006:475553 CAPLUS  
 DN 145:469446  
 TI Myelin-associated inhibitory molecules and immune therapy for spinal cord

regeneration  
 AU Yin, Guodong; Tang, Xun  
 CS Department of Orthopaedics, Kunming General Hospital of Chengdu Military Command, Kunming, 650032, Peop. Rep. China  
 SO Zhonghua Chuangshang Zazhi (2005), 21(7), 551-553  
 DN CODEN: ZCZAFD; ISSN: 1001-8050  
 TI Article  
 LA Chinese  
 ED Entered STN: 17 Jun 2003  
 Last Updated on STN: 17 Jun 2003

L14 ANSWER 17 OF 25 CAPLUS COPYRIGHT 2007 ACS on STN  
 AN 2006:267147 CAPLUS  
 DN 144:445304  
 TI Regeneration of lesioned entorhino-hippocampal axons in vitro by combined degradation of inhibitory proteoglycans and blockade of Nogo-66/NGR signaling  
 AU Mingorance, Ana; Sole, Marta; Muneton, Vilma; Martinez, Albert; Nieto-Sampedro, Manuel; Soriano, Eduardo; del Rio, Jose Antonio  
 CS Development and Regeneration of the Central Nervous System (CNS), Department of Cell Biology, Barcelona Science Park, University of Barcelona, Barcelona, 08028, Spain  
 SO PNAS (2006), 103(13), 491-493, 10.1096/fj.05-5121fje  
 DN CODEN: PNAS; ISSN: 0892-6638  
 TI Article  
 LA English  
 ED Entered STN: 17 Jun 2003  
 Last Updated on STN: 17 Jun 2003

L14 ANSWER 18 OF 25 CAPLUS COPYRIGHT 2007 ACS on STN  
 AN 2005:954635 CAPLUS  
 DN 143:283436  
 TI Why do Nogo/Nogo-66 receptor gene knockouts result in inferior regeneration compared to treatment with neutralizing agents?  
 AU Teng, Felicia Yu Hsuan; Tang, Bor Luen  
 CS Department of Biochemistry and Programme in Neurobiology and Aging, National University of Singapore, Singapore, Singapore, Singapore  
 SO Journal of Neurochemistry (2005), 94(4), 865-874  
 DN CODEN: JONRAG; ISSN: 0022-3042  
 TI Article  
 LA English  
 ED Entered STN: 17 Jun 2003  
 Last Updated on STN: 17 Jun 2003

L14 ANSWER 19 OF 25 CAPLUS COPYRIGHT 2007 ACS on STN  
 AN 2003:416397 CAPLUS  
 DN 139:332941  
 TI Delayed systemic Nogo-66 receptor antagonist promotes recovery from spinal cord injury  
 AU Li, Shuxin; Strittmatter, Stephen M.  
 CS Department of Neurology and Section of Neurobiology, Yale University School of Medicine, New Haven, CT, 06520, USA  
 SO Journal of Neuroscience (2003), 23(10), 4219-4227  
 DN CODEN: JNRSDS; ISSN: 0270-6474  
 TI Article  
 LA English  
 ED Entered STN: 17 Jun 2003  
 Last Updated on STN: 17 Jun 2003

L14 ANSWER 20 OF 25 CAPLUS COPYRIGHT 2007 ACS on STN  
 AN 2002:403264 CAPLUS  
 DN 137:362909

THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD  
 ALL CITATIONS AVAILABLE IN THE RE FORMAT

THERE ARE 71 CITED REFERENCES AVAILABLE FOR THIS RECORD  
 ALL CITATIONS AVAILABLE IN THE RE FORMAT

THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD  
 ALL CITATIONS AVAILABLE IN THE RE FORMAT

TI Nogo-66 receptor antagonist peptide promotes axonal regeneration  
AU GrandPre, Tadzia; Li, Shuxin; Strittmatter, Stephen M.  
CS Department of Neurology and Section of Neurobiology, Yale University  
School of Medicine, New Haven, CT, 06520, USA  
SO Nature (London, United Kingdom) (2002), 417(6888), 547-551  
CODEN: NATUAS; ISSN: 0028-0836  
PB Nature Publishing Group  
DT Journal  
LA English  
RE CNT 14 THERE ARE 14 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 21 OF 25 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN  
AN 2007183702 EMBASE  
TI Nogo-A is involved in secondary axonal degeneration of thalamus in  
hypertensive rats with focal cortical infarction.  
AU Wang F.; Liang Z.; Hou Q.; Xing S.; Ling L.; He M.; Pei Z.; Zeng J.  
CS J. Zeng, Department of Neurology, Stroke Center, The First Affiliated  
Hospital, No. 58, Zhongshan Road 2, Guangzhou, 510080, China.  
zengj@pub.guangzhou.gd.cn  
SO Neuroscience Letters, (7 May 2007) Vol. 417, No. 3, pp. 255-260.  
Refs: 18  
ISSN: 0304-3940 CODEN: NELED5  
PUI S 0304-3940(07)00245-5  
CY Ireland  
DT Journal; Article  
FS 029 Clinical Biochemistry  
037 Drug Literature Index  
005 General Pathology and Pathological Anatomy  
008 Neurology and Neurosurgery  
LA English  
SL English  
ED Entered STN: 31 May 2007  
Last Updated on STN: 31 May 2007

L14 ANSWER 22 OF 25 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN  
AN 2005378195 EMBASE  
TI Why do Nogo/Nogo-66 receptor gene knockouts result in inferior  
regeneration compared to treatment with neutralizing agents?  
AU Teng F.Y.H.; Tang B.L.  
CS B.L. Tang, Department of Biochemistry, Programme in Neurobiology and  
Aging, National University of Singapore, 8 Medical Drive, Singapore  
117597, Singapore. bchtbl@nus.edu.sg  
SO Journal of Neurochemistry, (2005) Vol. 94, No. 4, pp. 865-874.  
Refs: 71  
ISSN: 0022-3042 CODEN: JONRA  
CY United Kingdom  
DT Journal; (Short Survey)  
FS 008 Neurology and Neurosurgery  
022 Human Genetics  
037 Drug Literature Index  
LA English  
SL English  
ED Entered STN: 22 Sep 2005  
Last Updated on STN: 22 Sep 2005

L14 ANSWER 23 OF 25 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN  
AN 2005069346 EMBASE  
TI Strategies for developing Nogo antagonists.  
AU Prinjha R.K.; McAdam R.A.; Burbidge S.A.; Ellis J.H.  
CS R.K. Prinjha, Neurology and GI-CEDD, New Frontiers Science Park, Third

SO Drug Discovery Today: Therapeutic Strategies, (2004) Vol. 1, No. 1, pp. 21-27.  
Refs: 30  
ISSN: 1740-6773  
PUI S 1740-6773(04)00016-6  
CY United Kingdom  
DT Journal; Article  
FS 008 Neurology and Neurosurgery  
022 Human Genetics  
029 Clinical Biochemistry  
030 Pharmacology  
037 Drug Literature Index  
039 Pharmacy  
LA English  
SL English  
ED Entered STN: 24 Feb 2005  
Last Updated on STN: 24 Feb 2005

L14 ANSWER 24 OF 25 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN  
AN 200321746 EMBASE  
TI Delayed systemic Nogo-66 receptor antagonist promotes recovery from spinal cord injury.  
AU Li S.; Strittmatter S.M.  
CS Dr. S.M. Strittmatter, Department of Neurology, Section of Neurobiology,  
Yale University School of Medicine, P.O. Box 208018, New Haven, CT 06520,  
United States. stephen.strittmatter@yale.edu  
SO Journal of Neuroscience, (15 May 2003) Vol. 23, No. 10, pp. 4219-4227.  
Refs: 50  
ISSN: 0270-6474 CODEN: JNRSDS  
CY United States  
DT Journal; Article  
FS 008 Neurology and Neurosurgery  
037 Drug Literature Index  
LA English  
SL English  
ED Entered STN: 28 Aug 2003  
Last Updated on STN: 28 Aug 2003

L14 ANSWER 25 OF 25 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN  
AN 2002199201 EMBASE  
TI Nogo-66 receptor antagonist peptide promotes axonal regeneration.  
AU GrandPre T.; Shuxin L.I.; Strittmatter S.M.  
CS S.M. Strittmatter, Department of Neurology, Section of Neurobiology, Yale  
University School of Medicine, P.O. Box 208018, New Haven, CT 06520,  
United States. stephen.strittmatter@yale.edu  
SO Nature, (30 May 2002) Vol. 417, No. 6888, pp. 547-551.  
Refs: 14  
ISSN: 0028-0836 CODEN: NATUAS  
CY United Kingdom  
DT Journal; Article  
FS 008 Neurology and Neurosurgery  
037 Drug Literature Index  
LA English  
SL English  
ED Entered STN: 20 Jun 2002  
Last Updated on STN: 20 Jun 2002

=> s alzheimr and (axonal (w) regeneration)  
L17 63 ALZHEIMER AND (AXONAL (W) REGENERATION)

=> d 117 56-63

L17 ANSWER 58 OF 63 TOXCENTER COPYRIGHT 2007 ACS on STN  
AN 2005:193159 TOXCENTER  
CP Copyright 2007 ACS  
DN CA143142418060

TI A novel neurotrophic agent, T-817WA [1-[3-[2-(1-benzothiophen-5-yl)ethoxy]propyl]-3-azetidinol maleate], attenuates amyloid- $\beta$ -induced neurotoxicity and promotes neurite outgrowth in rat cultured central nervous system neurons

AU Hirata, Kazunari; Yanaguchi, Hidetoshi; Takamura, Yusaku; Takagi, Akiko; Fukushima, Tetsuo; Iwakami, Noboru; Saitoh, Akihito; Nakagawa, Masaya; Yanada, Tatsuo  
CS Research Laboratories, Toyama Chemical Co., Ltd., Toyama, Japan.  
SO Journal of Pharmacology and Experimental Therapeutics, (2005) Vol. 314, No. 1, pp. 252-259.  
CODEN: JPETAB. ISSN: 0022-3565.

CY JAPAN

DT Journal

FS CAPLUS

OS CAPLUS 2005:603092

LA English

ED Entered STN: 19 Jul 2005

Last Updated on STN: 29 Aug 2006

L17 ANSWER 59 OF 63 TOXCENTER COPYRIGHT 2007 ACS on STN

AN 2005:154827 TOXCENTER

CP Copyright (c) 2007 The Thomson Corporation

DN PREV200500203216

TI Neuroprotective role of testosterone in the nervous system

AU Bialek, Magdalena; Zaremba, Pawel; Borowicz, Kinga K.; Czuczwar, Stanislaw J. [Reprint Author]  
CS Dept Pathophysiol, Skubiszewski Med Univ, Jazczewskiego 8, PL-20090, Lublin, Poland czuczwar@yahoo.com

SO Polish Journal of Pharmacology, (September 2004) Vol. 56, No. 5, pp. 509-518. print.

ISSN: 1230-6002.

DT Article

FS General Review; (Literature Review)

OS BIOSIS 2005:211699

LA English

ED Entered STN: 7 Jun 2005

Last Updated on STN: 7 Jun 2005

L17 ANSWER 60 OF 63 TOXCENTER COPYRIGHT 2007 ACS on STN

AN 2005:92103 TOXCENTER

CP Copyright 2007 ACS

DN CA1421386101Z

TI Neuroprotective role of testosterone in the nervous system

AU Bialek, Magdalena; Zaremba, Pawel; Borowicz, Kinga K.; Czuczwar, Stanislaw J.  
CS Department of Pathophysiology, Skubiszewski Medical University, Lublin, PL 20-090, Pol.

SO Polish Journal of Pharmacology, (2004) Vol. 56, No. 5, pp. 509-518.

CODEN: RJPAE3. ISSN: 1230-6002.

CY POLAND

DT Journal

FS CAPLUS

OS CAPLUS 2005:243139

LA English

ED Entered STN: 22 Mar 2005

Last Updated on STN: 29 Nov 2005

L17 ANSWER 61 OF 63 TOXCENTER COPYRIGHT 2007 ACS on STN

AN 2003:55704 TOXCENTER

CP Copyright 2007 ACS

DN CA13812162764X

TI Recent advance in adenoviral gene transfer technology for neuronal survival and axonal regeneration

AU Namikawa, Kazuhiko; Kiyama, Hiroshi  
CS Dep. Anatomy, Grad. Sch. Med., Osaka City Univ., Japan.  
SO Sashin Igaku, (2002) Vol. 57, No. 7, pp. 1591-1600.

CODEN: SAIGAK. ISSN: 0370-8241.

CY JAPAN

DT Journal

FS CAPLUS

OS CAPLUS 2002:604210

LA Japanese

ED Entered STN: 11 Mar 2003

Last Updated on STN: 21 Feb 2006

L17 ANSWER 62 OF 63 TOXCENTER COPYRIGHT 2007 ACS on STN

AN 1997:28867 TOXCENTER

DN PubMed ID: 9017230

TI Postnatal retinal ganglion cells in vitro: protection against reactive

oxygen species (ROS)-induced axonal degeneration by cocultured astrocytes

AU Lucius R; Sievers J

CS Anatomisches Institut, Universitat Kiel, Germany

SO Brain research, (1996 Dec 16) Vol. 743, No. 1-2, pp. 56-62.

Journal code: 0045503. ISSN: 0006-8993.

CY Netherlands

DT Journal; Article; (JOURNAL ARTICLE)

FS (RESEARCH SUPPORT, NON-U.S. GOV'T)

OS MEDLINE 97169682

LA English

ED Entered STN: 16 Nov 2001

Last Updated on STN: 16 Nov 2001

L17 ANSWER 63 OF 63 TOXCENTER COPYRIGHT 2007 ACS on STN

AN 1996:218505 TOXCENTER

CP Copyright 2007 ACS

DN CA12607085782Z

TI Postnatal retinal ganglion cells in vitro: protection against reactive oxygen species (ROS)-induced axonal degeneration by cocultured astrocytes

AU Lucius, Ralph; Sievers, Jobst

CS Anatomisches Institut der Universitaet Kiel, Olshausenstr. 40, Kiel, D-24118, Germany.

SO Brain Research, (1996) Vol. 743, No. 1-2, pp. 56-62.

CODEN: BRREAP. ISSN: 0006-8993.

CY GERMANY, FEDERAL REPUBLIC OF

DT Journal

FS CAPLUS

OS CAPLUS 1996:736899

LA English

ED Entered STN: 16 Nov 2001

Last Updated on STN: 18 Jun 2002

=> d 117 1-63 kwic

L17 ANSWER 1 OF 63 MEDLINE on STN

AB The dogma that the adult central nervous system (CNS) is nonpermissive to axonal regeneration is beginning to fall in the face of increased understanding of the molecular and cellular biology of axon outgrowth. It . . . These vectors may be useful in regenerative strategies for spinal cord injury, brain injury, and neurodegenerative

diseases including Parkinson's disease, Alzheimer's disease, and Huntington's disease.

L17 ANSWER 2 OF 63 MEDLINE on STN

AB Injuries and diseases. After CNS injury, CSPGs are the major inhibitory component of the glial scar. Removal of CSPGs improves axonal regeneration and functional recovery. CSPGs may also be involved in the pathological processes in diseases such as epilepsy, stroke and Alzheimer's disease. Several possible methods of manipulating CSPGs in the CNS have recently been identified. The development of methods to remove.

L17 ANSWER 3 OF 63 MEDLINE on STN

AB Reticulon (RTN) proteins are localized to the endoplasmic reticulum (ER), and are related to intracellular membrane trafficking, apoptosis, inhibiting axonal regeneration, and Alzheimer's disease. The RTN proteins are produced without an N-terminal signal peptide. Their C-terminal domain contains two long hydrophobic segments. We.

L17 ANSWER 4 OF 63 MEDLINE on STN

AB Axons fail to regenerate in the adult central nervous system (CNS) following injury. Developing strategies to promote axonal regeneration is therapeutically attractive for various CNS pathologies such as traumatic brain injury, stroke and Alzheimer's disease. Because the RhoA pathway is involved in neurite outgrowth, Rho-associated kinases (ROCKs), downstream effectors of GTP-bound Rho, are potentially.

L17 ANSWER 5 OF 63 MEDLINE on STN

AB Progressive neuronal loss in Alzheimer's disease (AD) is considered to be a consequence of the neurotoxic properties of amyloid-beta peptides (A beta). T-817MA (1-{3-[2-(1-benzothienophen-5-yl) ethoxy]}-5-yl) agent for the treatment of AD based on its neuroprotective potency against A beta-induced neurotoxicity and its effect of enhancing axonal regeneration in the sciatic nerve axotomy model. The neuroprotective effect of T-817MA against A beta(1-42) or oxidative stress-induced neurotoxicity was assessed.

L17 ANSWER 6 OF 63 MEDLINE on STN

AB Testosterone actions is neuroprotection. There are some evidences supporting the hypothesis that testosterone may act protectively in neurodegenerative disorders, e.g. Alzheimer's disease (AD), mild cognitive impairment (MCI) or depression. Androgens alter also the morphology, survival and axonal regeneration of motor neurons. These hormones accelerate the regeneration of hamster facial nerve and anterior tibialis sciatic nerve in rabbits following.

L17 ANSWER 7 OF 63 MEDLINE on STN

TI Modulation of axonal regeneration in neurodegenerative disease: focus on Nogo.

AB Recent work has demonstrated that axonal regeneration in the central nervous system is limited by myelin-derived Nogo binding to an axonal Nogo Receptor. The Nogo system appears to be a physiologic role in regulating structural plasticity. The possibility that the Nogo system contributes to pathologic and compensatory plasticity in Alzheimer's disease is considered.

CT Alzheimer Disease: ME, metabolism  
Alzheimer Disease: PA, pathology

Animals

\*Axons: ME, metabolism

\*Axons: PA, pathology

\*Growth Inhibitors: ME, metabolism

\*Myelin Proteins: ME, metabolism

Myelin.

L17 ANSWER 8 OF 63 MEDLINE on STN

AB Estrogen in neuroprotection. Accumulated clinical evidence suggests that estrogen exposure decreases the risk and delays the onset and progression of Alzheimer's disease and schizophrenia, and may also enhance recovery from traumatic neurological injury such as stroke. Recent basic science studies show the classical nuclear estrogen receptor, through which estrogen alters expression of estrogen responsive genes that play a role in apoptosis, axonal regeneration, or general trophic support. Yet another possibility is that estrogen receptors in the membrane or cytoplasm alter phosphorylation cascades through.

L17 ANSWER 9 OF 63 MEDLINE on STN

AB Reactive oxygen species (ROS) are supposed to be involved in neurodegenerative processes like Parkinson's or Alzheimer's disease. Beside this there are an increasing number of studies indicating an involvement of ROS in traumatic brain injury. We astrocytes are able to protect retinal ganglion cells against ROS-induced oxidative stress, (ii) astrocytes release soluble neurotrophic factors supporting RGC axonal regeneration, and (iii) free radical production after tissue injury may partly contribute to the failure of axonal regeneration in the adult mammalian central nervous system.

L17 ANSWER 10 OF 63 MEDLINE on STN

TI Aberrant GAP-43 gene expression in Alzheimer's disease. GAP-43 is a growth-associated phosphoprotein expressed at high levels in neurons during development, axonal regeneration, and neurite sprouting. GAP-43 gene expression in mature neurons is probably functionally important for the structural remodeling of synapses as required for learning and establishing new memory. The widespread aberrant neurite growth accompanied by impaired synaptic plasticity in Alzheimer's disease (AD) suggests that abnormal GAP-43 gene expression may contribute to the cascade of neurodegeneration. In the present study, end-stage.

CT Check Tags: Female; Male

Aged, 80 and over

Aging: ME, metabolism

\*Alzheimer Disease: GE, genetics

\*Alzheimer Disease: PA, pathology

Blotting, Northern

Brain: ME, metabolism

Brain: PA, pathology

GAP-43 Protein

\*Gene Expression

Humans

Immunohistochemistry

In Situ.

L17 ANSWER 11 OF 63 MEDLINE on STN

AB Possible molecular techniques underlying nerve growth are discussed. Possible therapeutic approaches are presented for many neurologic disorders, ranging from stroke to Alzheimer's disease to acquired immunodeficiency syndrome, based on regrowing or saving injured neurons. The clinical neurologist will become important in practical applications and research into prolonging neuronal survival and fostering axonal regeneration. Over the coming years, with further research, it is anticipated that patients will be treated with these or similar modulatory.

L17 ANSWER 12 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on

STN  
 AB apoptosis, inhibiting axonal regeneration, and Alzheimer's disease. The RTN proteins are produced without an N-terminal signal peptide. Their C-terminal domain contains two long hydrophobic segments. We.

L17 ANSWER 13 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN  
 AB Axons fail to regenerate in the adult central nervous system (CNS) following injury. Developing strategies to promote axonal regeneration is therapeutically attractive for various CNS pathologies such as traumatic brain injury, stroke and Alzheimer's disease. Because the RhoA pathway is involved in neurite outgrowth, Rho-associated kinases (ROCKs), downstream effectors of GTP-bound Rho, are potentially.

IT Brain injury: nervous system disease, injury  
 Brain injuries (MeSH)

IT Diseases  
 stroke: vascular disease, nervous system disease  
 Cerebrovascular Disorders (MeSH)

IT Diseases  
 Alzheimer's disease: nervous system disease; behavioral and mental disorders

IT Chemicals & Biochemicals  
 RhoA; ROCK; Y-27632: enzyme inhibitor-drug; cofilin: dephosphorylation; H-1152.

L17 ANSWER 14 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN  
 AB Progressive neuronal loss in Alzheimer's disease (AD) is considered to be a consequence of the neurotoxic properties of amyloid-beta peptides (A beta). T-817MA (1-{3-[2-(1-benzothiophen-5-yl)-1-hydroxy-2-oxo-1,2,3,4-tetrahydro-1H-benz[e][1,2,4]oxadiazol-5-yl]propan-1-yl}pyrrolidine-2-carboxamide) is a potent inhibitor of A beta-induced neurotoxicity and its effect of enhancing axonal regeneration in the sciatic nerve axotomy model. The neuroprotective effect of T-817MA against A beta(1-42) or oxidative stress-induced neurotoxicity was assessed.

IT of Organisms  
 neuron: nervous system; central nervous system: nervous system; glial cell: nervous system; cortical neuron: nervous system

IT Diseases  
 Alzheimer's disease: nervous system disease, behavioral and mental disorders

IT Chemicals & Biochemicals  
 hydrogen peroxide; growth-associated protein 43; GSH; amyloid-beta: toxin.

L17 ANSWER 15 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN  
 AB testosterone actions is neuroprotection. There are some evidences supporting the hypothesis that testosterone may act protectively in neurodegenerative disorders, e.g. Alzheimer's disease (AD), mild cognitive impairment (MCI) or depression. Androgens alter also the morphology, survival and axonal regeneration of motor neurons. These hormones accelerate the regeneration of hamster facial nerve and anterior tibialis sciatic nerve in rabbits following.

IT nervous system; laryngeal motor nucleus: nervous system; motor neuron: nervous system; pelvic autonomic neuron: nervous system; spinal cord

IT Diseases  
 Alzheimer's disease: behavioral and mental disorders, nervous system disease, drug therapy

IT Diseases  
 Alzheimer Disease (MeSH)  
 Depression (MeSH)  
 Diseases  
 mild cognitive impairment: behavioral and mental.

L17 ANSWER 16 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN  
 TI Modulation of axonal regeneration in neurodegenerative disease: Focus on Nogo.  
 AB Recent work has demonstrated that axonal regeneration in the central nervous system is limited by myelin-derived Nogo binding to an axonal Nogo receptor. The Nogo system appears to be a physiologic role in regulating structural plasticity. The possibility that the Nogo system contributes to pathologic and compensatory plasticity in Alzheimer's Disease is considered.

IT Structures, & Systems of Organisms  
 axon: nervous system, plasticity, regeneration; central nervous system: nervous system; neurite: nervous system

IT Diseases  
 Alzheimer's disease: behavioral and mental disorders, nervous system disease

IT Chemicals & Biochemicals  
 Nogo: binding activity; Nogo receptor; myelin

L17 ANSWER 17 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN  
 AB individuals with moderate or severe white matter changes (WMC) and in those with mild or no WMC. Twenty-two patients with Alzheimer's disease (AD), nine patients with subcortical vascular dementia (SVD), and 20 normal controls were included in the study. The occurrence.

IT Medicine, Medical Sciences)

IT Parts, Structures, & Systems of Organisms  
 cerebrospinal fluid: nervous system; white matter: nervous system

IT Diseases  
 Alzheimer's disease: behavioral and mental disorders, nervous system disease

IT Diseases  
 Alzheimer Disease (MeSH)  
 subcortical vascular dementia: behavioral and mental disorders  
 Chemicals & Biochemicals  
 beta-amyloid 42: neurofilament protein; tau:  
 Miscellaneous Descriptors  
 apolipoprotein E E4 allele inheritance; axonal regeneration; white matter changes

L17 ANSWER 18 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN  
 IT Major Concepts  
 Nervous System (Neural Coordination); Pharmacology  
 IT Parts, Structures, & Systems of Organisms  
 brain: nervous system

IT Diseases  
 Alzheimer's type senile dementia: behavioral and mental disorders, nervous system disease

IT Chemicals & Biochemicals  
 TPI-3356 [(16S)-15-deoxy-16-hydroxy-16-methyl-9(O)-methano-delta-6(9alpha)-prostaglandin]: anti-amnesic effect, prostacyclin stable.

IT Miscellaneous Descriptors



axonal regeneration; learning; memory

- L17 ANSWER 19 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN  
AB Reactive oxygen species (ROS) are supposed to be involved in neurodegenerative processes like Parkinson's or Alzheimer's disease. Beside this there are an increasing number of studies indicating an involvement of ROS in traumatic brain injury. We... astrocytes are able to protect retinal ganglion cells against ROS-induced oxidative stress, (ii) astrocytes release soluble neurotrophic factors supporting axonal regeneration, and (iii) free radical production after tissue injury may partly contribute to the failure of axonal regeneration in the adult mammalian central nervous system.
- L17 ANSWER 20 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN  
TI Aberrant GAP-43 gene expression in Alzheimer's disease.  
AB GAP-43 is a growth-associated phosphoprotein expressed at high levels in neurons during development, axonal regeneration, and neuritic sprouting. GAP-43 gene expression in mature neurons is probably functionally important for the structural remodeling of synapses as required for learning and establishing new memory. The widespread aberrant neuritic growth accompanied by impaired synaptic plasticity in Alzheimer's disease (AD) suggests that abnormal GAP-43 gene expression may contribute to the cascade of neurodegeneration. In the present study, end-stage.
- L17 ANSWER 21 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN  
TI Disrupted beta-1-adrenoceptor-G protein coupling in the temporal cortex of patients with Alzheimer's disease.  
AB... The efficacy of beta-1-adrenoceptor-G protein coupling was studied in postmortem temporal cortex synaptic membranes from a series of control and Alzheimer's disease subjects. For the control cases, the non-hydrolyzable GTP analogue 5'-guanylimidodiphosphate (Gpp(NH)p) gave a significant reduction in the affinity of... effect was attributed to the conversion of high agonist-affinity sites to a lower-affinity state and was not found for the Alzheimer's disease cases. These data indicate that a disruption of beta-1-adrenoceptor-G protein coupling occurs in the temporal cortex of Alzheimer's disease patients.
- IT Miscellaneous Descriptors  
AXONAL REGENERATION IMPAIRMENT; HYPEREMIA;  
HYPERGLYCEMIA; SENSORY CONDUCTION VELOCITY; VASA NERVORUM NEUROGENIC INFLAMMATION
- L17 ANSWER 22 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN  
TI... The carboxy-terminus of the beta amyloid protein is critical for the seeding of amyloid formation: Implications for the pathogenesis of Alzheimer's disease.  
AB... beta-1-40, beta-1-42, and beta-1-43), have been identified as the major components of the cerebral amyloid deposits which are characteristic of Alzheimer's disease. Kinetic studies of aggregation by three naturally occurring beta protein variants (beta-1-39, beta-1-40, beta-1-42) and four model peptides (beta-26-39).
- IT Miscellaneous Descriptors  
AXONAL REGENERATION; CYCLOSPORIN A;  
IMMUNOSUPPRESSANT-DRUG; NERVE ANASTOMOSIS; NERVE REPAIR; NERVE TRANSPLANTATION; PERINEURIUM
- L17 ANSWER 23 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN  
AB... axotomized medial septal and diagonal band of Broca neurons

selectively and rapidly express JLI. The role of Jun expression in axonal regeneration or neuronal death is discussed.

- IT Miscellaneous Descriptors  
RAT AXONAL REGENERATION NEURONAL DEATH PROTEIN  
ALZHEIMER'S DISEASE TRANSCRIPTION FACTORS
- L17 ANSWER 24 OF 63 CAPLUS COPYRIGHT 2007 ACS on STN  
AB... injuries and diseases. After CNS injury, CSPGs are the major inhibitory component of the glial scar. Removal of CSPGs improves axonal regeneration and functional recovery. CSPGs may also be involved in the pathol. processes in diseases such as epilepsy, stroke and Alzheimer's disease. Several possible methods of manipulating CSPGs in the CNS have recently been identified. The development of methods to remove.
- L17 ANSWER 25 OF 63 CAPLUS COPYRIGHT 2007 ACS on STN  
AB... certain polypeptides and polypeptide fragments of Nogo receptor-1 (NGR1) and Nogo receptor-2 (NGR2) for promoting neurite outgrowth, neuronal survival, and axonal regeneration in CNS neurons. Previous studies have shown that the entire leucine rich repeat (LRR) region of NGR1, including the C-terminal. Typically, the polypeptides and polypeptide fragments of the invention act to block NGR-mediated inhibition of neuronal survival, neurite outgrowth or axonal regeneration of CNS (central nervous system) neurons by inhibiting signal transduction by the NGR complex. Nogo receptor NGR1 NGR2 disulfide structure mutant neurite outgrowth; NGR Nogo receptor signaling inhibition CNS neuron axonal regeneration
- IT Alzheimer's disease  
Axon  
Central nervous system agents  
Disulfide group  
Gene therapy  
Glioma (disease)  
Hearing loss  
Human  
Mammalia  
Molecular cloning  
Multiple sclerosis  
Parkinson's disease  
(Nogo receptor (NGR) disulfide structure, NGR signaling inhibiting NGR fragments, mutants, fusion products and genetic constructs, and uses in mediating axonal growth)  
Central nervous system  
(neurons, promoting axonal regeneration in; Nogo receptor (NGR) disulfide structure, NGR signaling inhibiting NGR fragments, mutants, fusion products and genetic constructs, and uses in mediating axonal growth)
- L17 ANSWER 26 OF 63 CAPLUS COPYRIGHT 2007 ACS on STN  
AB Reticulon (RTN) proteins are localized to the endoplasmic reticulum (ER), and are related to intracellular membrane trafficking, apoptosis, inhibiting axonal regeneration, and Alzheimer's disease. The RTN proteins are produced without an N-terminal signal peptide. Their C-terminal domain contains two long hydrophobic segments. We.
- L17 ANSWER 27 OF 63 CAPLUS COPYRIGHT 2007 ACS on STN  
AB Axons fail to regenerate in the adult central nervous system (CNS) following injury. Developing strategies to promote axonal regeneration is therapeutically attractive for various CNS pathologies such as traumatic brain injury, stroke and Alzheimer's disease. Because the RhoA pathway is involved in neurite outgrowth, Rho-associated kinases (ROCKs), downstream effectors of GTP-bound Rho, are

potentially.

LI7 ANSWER 28 OF 63 CAPLUS COPYRIGHT 2007 ACS on STN  
 AB . . . may be used as antagonists to Ngr1 ligands and, as such, may be useful in treating subjects in need of axonal regeneration (e.g., for antagonizing (e.g., reversing, decreasing, reducing, preventing, etc.) axonal growth inhibition mediated by such Ngr1 ligands, and for screening Alzheimer's disease Multiple sclerosis Parkinson's disease (treating, Nogo receptor 1 (Ngr1) functional motifs and peptide mimetics and use as antagonists to Ngr1 ligands for antagonizing axonal growth inhibition)

IT IT

LI7 ANSWER 29 OF 63 CAPLUS COPYRIGHT 2007 ACS on STN  
 TI Protein sequences of human, mouse and chicken netrin-G1 ligand NGL-1 and uses in promotion of axonal regeneration  
 AB Described are the protein sequences of human, mouse and chicken netrin-G1 ligand NGL-1 and their uses in promoting axonal regeneration. The invention concerns a method of promoting the growth or regeneration of neurons, and treating disease or conditions associated with.

IT IT

IT Protein motifs (Ig-like domain, of NGL-1; protein sequences of human, mouse and chicken netrin-G1 ligand NGL-1 and uses in promotion of axonal regeneration)

IT IT

IT Antibodies and immunoglobulins (against NGL-1; protein sequences of human, mouse and chicken netrin-G1 ligand NGL-1 and uses in promotion of axonal regeneration)

IT IT

IT Antibodies and immunoglobulins (chimeric, against NGL-1; protein sequences of human, mouse and chicken netrin-G1 ligand NGL-1 and uses in promotion of axonal regeneration)

IT IT

IT Antibodies and immunoglobulins (humanized, against NGL-1; protein sequences of human, mouse and chicken netrin-G1 ligand NGL-1 and uses in promotion of axonal regeneration)

IT IT

IT Proteins (BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses) (leucine-rich repeat, NGL-1 (netrin-G1 ligand 1); protein sequences of human, mouse and chicken netrin-G1 ligand NGL-1 and uses in promotion of axonal regeneration)

IT IT

IT Repeat motifs (protein) (leucine-rich repeat, of NGL-1; protein sequences of human, mouse and chicken netrin-G1 ligand NGL-1 and uses in promotion of axonal regeneration)

IT IT

IT Proteins (BSU (Biological study, unclassified); BIOL (Biological study) (netrin, G1; protein sequences of human, mouse and chicken netrin-G1

IT IT

IT Molecular association (netrin-G1 binding to NGL-1; protein sequences of human, mouse and chicken netrin-G1 ligand NGL-1 and uses in promotion of axonal regeneration)

IT IT

IT Signal peptides (of NGL-1; protein sequences of human, mouse and chicken netrin-G1 ligand NGL-1 and uses in promotion of axonal regeneration)

IT IT

IT Axon (outgrowth; protein sequences of human, mouse and chicken netrin-G1 ligand NGL-1 and uses in promotion of axonal regeneration)

IT IT

IT Analgesics Anti-Alzheimer's agents Antiarteriosclerotics Antiparkinsonian agents (protein sequences of human, mouse and chicken netrin-G1 ligand NGL-1 and uses in nerve cell dysfunction)

IT IT

IT Mus musculus Human Protein sequences (protein sequences of human, mouse and chicken netrin-G1 ligand NGL-1 and uses in promotion of axonal regeneration)

IT IT

IT Axon (regeneration; protein sequences of human, mouse and chicken netrin-G1 ligand NGL-1 and uses in promotion of axonal regeneration)

IT IT

IT Alzheimer's disease Multiple sclerosis Parkinson's disease (treatment of; protein sequences of human, mouse and chicken netrin-G1 ligand NGL-1 and uses in nerve cell dysfunction)

IT IT

IT 875208-52-9 (Biological study) (PDZ domain-binding motif of NGL-1; -protein sequences of human, mouse and chicken netrin-G1 ligand NGL-1 and uses in promotion of axonal regeneration)

IT IT

IT 875717-48-9D, subfragment is claimed 875717-49-0 875717-50-3 RL: BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses) (amino acid sequence; protein sequences of human, mouse and chicken netrin-G1 ligand NGL-1 and uses in promotion of axonal regeneration)

IT IT

IT 875717-66-1 875717-67-2 875717-68-3 RL: PRP (Properties) (unclaimed protein sequence; protein sequences of human, mouse and chicken netrin-G1 ligand NGL-1 and uses in promotion of axonal regeneration)

IT IT

IT 875612-87-6 RL: PRP (Properties) (unclaimed sequence; protein sequences of human, mouse and chicken netrin-G1 ligand NGL-1 and uses in promotion of axonal regeneration)

LI7 ANSWER 30 OF 63 CAPLUS COPYRIGHT 2007 ACS on STN  
 AB . . . for central nervous system repair, focusing on the therapeutic use of growth factors to reduce cell loss and to enhance axonal regeneration in the context of both neurodegenerative and traumatic disorders.

ST review NGF axon nerve regeneration CNS Alzheimer disease

IT Nervous system, disease  
(Huntington's chorea; growth factor can be used to reduce cell loss and to enhance axonal regeneration in animal model with Huntington's disease)

IT Nervous system, disease  
(amyotrophic lateral sclerosis; growth factor can be used to reduce cell loss and to enhance axonal regeneration in animal model with amyotrophic lateral sclerosis)

IT Alzheimer's disease  
(growth factor can be used to reduce cell loss and to enhance axonal regeneration in animal model with Alzheimer's disease)

IT Parkinson's disease  
(growth factor can be used to reduce cell loss and to enhance axonal regeneration in animal model with Parkinson's disease)

IT Multiple sclerosis  
(growth factor can be used to reduce cell loss and to enhance axonal regeneration in animal model with multiple sclerosis)

IT Brain  
Nerve regeneration  
(growth factor can be used to reduce cell loss and to enhance axonal regeneration in animal model with neurodegenerative and traumatic disorders)

IT Spinal cord, disease  
(injury; growth factor can be used to reduce cell loss and to enhance axonal regeneration in animal model with spinal cord injury)

IT Injury  
(spinal cord; growth factor can be used to reduce cell loss and to enhance axonal regeneration in animal model with spinal cord injury)

IT 9061-61-4, Nerve growth factor  
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
(growth factor can be used to reduce cell loss and to enhance axonal regeneration in animal model with neurodegenerative and traumatic disorders)

L17 ANSWER 31 OF 63 CAPLUS COPYRIGHT 2007 ACS on STN  
AB Progressive neuronal loss in Alzheimer's disease (AD) is considered to be a consequence of the neurotoxic properties of amyloid- $\beta$  peptides (A $\beta$ ). T-817MA was screened as a therapeutic agent for the treatment of AD based on its neuroprotective potency against A $\beta$ -induced neurotoxicity and its effect of enhancing axonal regeneration in the sciatic nerve axotomy model. The neuroprotective effect of T-817MA against A $\beta$  (1-42) or oxidative stress-induced neurotoxicity was assessed using.

IT Alzheimer's disease  
Anti-Alzheimer's agents  
Oxidative stress, biological  
(neuroprotective effects of T-817MA against  $\beta$ -amyloid- and oxidative stress-induced neurotoxicity in rat cultured central nervous system neurons)

L17 ANSWER 32 OF 63 CAPLUS COPYRIGHT 2007 ACS on STN  
AB ... neurogenesis, neuronal growth and regeneration, neuronal survival, and synaptic plasticity. Like neurotrophic factors, lithium and valproate promote neurite outgrowth and axonal regeneration in cultured neuronal cells and in injury models utilizing retinal cells, sciatic nerve, and spinal cord. These mood stabilizers also. . . protect cultured cells against a variety of

insults and reduce neuronal loss and associated functional deficits in animal models of Alzheimer's disease, HIV-associated encephalitis and dementia, Huntington's disease, ischemia, and Parkinson's disease. Cross-sectional and longitudinal brain imaging studies show that lithium.

Disease models  
(lithium, valproate showed neurotrophic action by reducing neuronal loss, associated functional deficit via ERK, PI3K pathway in animal model of Alzheimer's, Parkinson's, Huntington's disease, ischemia, HIV associated encephalitis, dementia)

IT Alzheimer's disease  
(mood stabilizer lithium and valproate showed neurotrophic action by reducing neuronal loss and associated functional deficit via ERK, PI3K pathway activation in animal model of Alzheimer's disease).

IT Brain  
(mood stabilizer lithium, valproate promoted neurogenesis, axonal regeneration, reduced neuronal loss in animal model and increased brain N-acetyl aspartate, cerebral gray matter via ERK, PI3K pathway activation in mood disorder patient)

IT Neuron  
(mood stabilizer lithium, valproate promoted neurogenesis, neurite outgrowth and axonal regeneration via ERK, PI3K pathway activation in cultured neuronal cell)

IT Nervous system agents  
(mood stabilizer; lithium, valproate promoted neurogenesis, axonal regeneration, reduced neuronal loss in animal model and increased brain N-acetyl aspartate, cerebral gray matter via ERK, PI3K pathway activation in mood disorder patient)

IT 115926-52-8, Phosphoinositide 3-Kinase 142243-02-5, Extracellular signal regulated kinase  
RL: BSU (Biological study, unclassified); BIOL (Biological study)  
(mood stabilizer lithium, valproate promoted neurogenesis, axonal regeneration, reduced neuronal loss in animal model and increased brain N-acetyl aspartate, cerebral gray matter via ERK, PI3K pathway activation in mood disorder patient)

IT 99-66-1 7439-93-2D, Lithium, salts  
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
(mood stabilizer lithium, valproate promoted neurogenesis, axonal regeneration, reduced neuronal loss in animal model and increased brain N-acetyl aspartate, cerebral gray matter via ERK, PI3K pathway activation in mood disorder patient)

L17 ANSWER 33 OF 63 CAPLUS COPYRIGHT 2007 ACS on STN  
AB . . . testosterone actions is neuroprotection. There is some evidence supporting the hypothesis that testosterone may act protectively in neurodegenerative disorders, e.g. Alzheimer's disease (AD), mild cognitive impairment (MCI), or depression. Androgens alter also the morphol., survival and axonal regeneration of motor neurons. These hormones accelerate the regeneration of hamster facial nerve and anterior tibialis sciatic nerve in rabbits following.

L17 ANSWER 34 OF 63 CAPLUS COPYRIGHT 2007 ACS on STN  
TI Modulation of axonal regeneration in neurodegenerative disease. Focus on Nogo  
AB A review. Recent work has demonstrated that axonal regeneration in the central nervous system is limited by myelin-derived Nogo binding to an axonal Nogo Receptor. The Nogo system appears. . . a physiol. role in regulating structural plasticity. The possibility that the Nogo system contributes to pathol. and compensatory plasticity in Alzheimer's disease is considered.  
ST review Nogo receptor axon regeneration neurodegeneration Alzheimer  
IT Alzheimer's disease  
Axon

IT Nerve regeneration  
Nerve regeneration  
Synaptic plasticity  
(Nogo receptor in modulation of axonal regeneration in neurodegenerative disease)

IT Proteins  
Receptors  
RL: BSU (Biological study, unclassified); BIOL (Biological study)  
RL: Nogo; Nogo receptor in modulation of axonal regeneration in neurodegenerative disease

IT Nervous system, disease  
(degeneration; Nogo receptor in modulation of axonal regeneration in neurodegenerative disease)

L17 ANSWER 35 OF 63 CAPLUS COPYRIGHT 2007 ACS on STN  
TI Recent advance in adenoviral gene transfer technology for neuronal survival and axonal regeneration

AB A review. Neuron-targeted gene transfer by adenovirus for the gene therapy of neuronal survival and axonal regeneration in the treatment of Parkinson's disease, Alzheimer's disease, malignant glioma etc. is reviewed.

IT Nerve regeneration  
(axonal; recent advance in adenoviral gene transfer technol. for neuronal survival and axonal regeneration)

IT Antitumor agents  
(glioma; recent advance in adenoviral gene transfer technol. for neuronal survival and axonal regeneration)

IT Adenoviral vectors  
Alzheimer's disease  
Anti-Alzheimer's agents  
Antiparkinsonian agents  
Gene therapy  
Neuroglia, neoplasm  
Parkinson's disease  
Transformation, genetic  
(recent advance in adenoviral gene transfer technol. for neuronal survival and axonal regeneration)

L17 ANSWER 36 OF 63 CAPLUS COPYRIGHT 2007 ACS on STN  
TI Apolipoprotein E and lipid mobilization in neuronal membrane remodeling and its relevance to Alzheimer's disease

AB A review on the link between apolipoprotein E (apoE) to either one of the two hallmarks of Alzheimer's disease (AD), namely amyloid plaque formation and neurofibrillary tangles. It includes a description of apolipoprotein E (apoE) and its gene... the role of apoE as a modulator of lipid homeostasis and synaptic plasticity. The well established peripheral nerve model of axonal regeneration and remyelination involving apoE and LDL receptors is presented first. The entorhinal cortex lesioning (ECL) model, which mimics certain neuropathol...  
neuron regeneration  
Gene, animal  
RL: BSU (Biological study, unclassified); BIOL (Biological study)  
(APOE; apolipoprotein E and lipid mobilization in neuronal membrane remodeling and its relevance to Alzheimer's disease)

IT Apolipoproteins  
RL: BSU (Biological study, unclassified); BIOL (Biological study)  
(E; apolipoprotein E and lipid mobilization in neuronal membrane remodeling and its relevance to Alzheimer's disease)

IT Lipoprotein receptors  
RL: BSU (Biological study, unclassified); BIOL (Biological study)  
(LDL; apolipoprotein E and lipid mobilization in neuronal membrane remodeling and its relevance to Alzheimer's disease)

IT Human  
Nerve regeneration  
Neurofibrillary tangle  
Synaptic plasticity  
(apolipoprotein E and lipid mobilization in neuronal membrane remodeling and its relevance to Alzheimer's disease)

IT Brain  
(entorhinal cortex; apolipoprotein E and lipid mobilization in neuronal membrane remodeling and its relevance to Alzheimer's disease)

IT Brain  
(hippocampus; apolipoprotein E and lipid mobilization in neuronal membrane remodeling and its relevance to Alzheimer's disease)

L17 ANSWER 37 OF 63 CAPLUS COPYRIGHT 2007 ACS on STN  
AB ... estrogen in neuroprotection. Accumulated clin. evidence suggests that estrogen exposure decreases the risk and delays the onset and progression of Alzheimer's disease and schizophrenia, and may also enhance recovery from traumatic neural injury such as stroke. Recent basic science studies show... the classical nuclear estrogen receptor, through which estrogen alters expression of estrogen responsive genes that play a role in apoptosis, axonal regeneration, or general trophic support. Yet another possibility is that estrogen receptors in the membrane or cytoplasm alter phosphorylation cascades through...  
Anti-Alzheimer's agents  
Cognition enhancers  
Schizophrenia  
(neuroprotection by estradiol and involved mechanisms)

L17 ANSWER 38 OF 63 CAPLUS COPYRIGHT 2007 ACS on STN  
AB ... Immediately following the lesioning, an osmotic pump to deliver serum complement and anti-galactocerebroside IGC was implanted at T11. Subsequently, brainstem-spinal axonal regeneration was observed in exptl. animals, as assessed by retrograde neuronal labeling with Fluorogold.

IT Alzheimer's disease  
Parkinson's disease  
(complement-dependent antibody-mediated transient demyelination for promotion of neuronal regrowth and regeneration in)

L17 ANSWER 39 OF 63 CAPLUS COPYRIGHT 2007 ACS on STN  
AB Reactive oxygen species (ROS) are supposed to be involved in neurodegenerative processes like Parkinson's or Alzheimer's disease. Beside this there are an increasing number of studies indicating an involvement of ROS in traumatic brain injury. We astrocytes are able to protect retinal ganglion cells against ROS-induced oxidative stress, (ii) astrocytes release soluble neurotrophic factors supporting RGC axonal regeneration, and (iii) free radical production after tissue injury may partly contribute to the failure of axonal regeneration in the adult mammalian central nervous system.

IT Alzheimer's disease  
Astrocyte  
Oxidative stress, biological  
Parkinson's disease  
(astrocytes against neurotoxic effects of reactive oxygen species in cocultures of cortical astrocytes with regenerating postnatal retinal ganglion cells)

L17 ANSWER 40 OF 63 CAPLUS COPYRIGHT 2007 ACS on STN  
TI Aberrant GAP-43 gene expression in Alzheimer's disease  
AB GAP-43 is a growth-associated phosphoprotein expressed at high levels in

neurons during development, axonal regeneration, and neurite sprouting. GAP-43 gene expression in mature neurons is probably functionally important for the structural remodeling of synapses as required for learning and establishing new memory. The widespread aberrant neurite growth accompanied by impaired synaptic plasticity in Alzheimer's disease (AD) suggests that abnormal GAP-43 gene expression may contribute to the cascade of neurodegeneration. In the present study, end-stage.

ST Gene GAP43 brain Alzheimer

IT Brain

Neuroglia  
(GAP-43 gene expression in human brain in Alzheimer's disease)

IT Gene, animal

RL: BSU (Biological study, unclassified); BIOL (Biological study)  
(GAP-43 gene expression in human brain in Alzheimer's disease)

IT Mental disorder  
(Alzheimer's disease, GAP-43 gene expression in human brain in Alzheimer's disease)

IT Phospholipoproteins  
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative); OCCU (Occurrence)  
(B-50, GAP-43 gene expression in human brain in Alzheimer's disease)

IT Nerve, disease  
(degeneration, GAP-43 gene expression in human brain in Alzheimer's disease)

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AB . . . injuries and diseases. After CNS injury, CSPGs are the major inhibitory component of the glial scar. Removal of CSPGs improves axonal regeneration and functional recovery. CSPGs may also be involved in the pathological processes in diseases such as epilepsy, stroke and Alzheimer's disease. Several possible methods of manipulating CSPGs in the CNS have recently been identified. The development of methods to remove.

CT Medical Descriptors:

Alzheimer disease: ET, etiology

epilepsy: ET, etiology

eye dominance

glia

human

hypothalamus hypophysis system

learning

memory

nerve cell lesion

\*nerve cell plasticity

\*nerve fiber regeneration

nonhuman

priority journal

review

stroke: ET, etiology

aggreacan: EC, endogenous.

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AB . . . evidence of developmental plasticity in these ex vivo models, demonstrating emergence of injury-stimulated neuronal progenitor cells, and neurite sprouting and axonal regeneration following pathway lesioning. Neuro- and axo-genesis are emerging as significant factors contributing to brain repair following many acute and

chronic.

CT Medical Descriptors:

Alzheimer disease: ET, etiology

Parkinson disease: ET, etiology

acute disease

amyotrophic lateral sclerosis: DT, drug therapy

anoxia

brain function

brain injury

cell assay

cell function

cell viability

chronic disease

clinical trial

coculture

\*degenerative.

L17 ANSWER 43 OF 63 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

AB Reticulon (RTN) proteins are localized to the endoplasmic reticulum (ER), and are related to intracellular membrane trafficking, apoptosis, inhibiting axonal regeneration, and Alzheimer's disease. The RTN proteins are produced without an N-terminal signal peptide. Their C-terminal domain contains two long hydrophobic segments. We.

CT Medical Descriptors:

Alzheimer disease

Golgi complex

amino terminal sequence

animal cell

article

carboxy terminal sequence

cellular distribution

\*endoplasmic reticulum

hydrophobicity

membrane transport

nerve fiber regeneration

nonhuman

nucleotide sequence

priority journal

protein determination

protein domain

protein family

protein localization

\*cell protein:..

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AB Axons fail to regenerate in the adult central nervous system (CNS) following injury. Developing strategies to promote axonal regeneration is therapeutically attractive for various CNS pathologies such as traumatic brain injury, stroke and Alzheimer's disease. Because the RhoA pathway is involved in neurite outgrowth, Rho-associated kinases (ROCKs), downstream effectors of GTP-bound Rho, are potentially.

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AB . . . interest. Studies focusing on animal and human olfactory bulb ensheathing cells (OECs) have heightened the expectations that OECs can enhance axonal regeneration and repair demyelinating diseases. Harvest of OECs from the olfactory bulb requires highly invasive surgery, which is a major obstacle. . . .

- CT Medical Descriptors:  
 \*olfactory . . . regeneration  
 demyelinating disease: TH, therapy  
 olfactory epithelium  
 nerve cell  
 culture technique  
 cell population  
 mitosis  
 autologous stem cell transplantation  
 donor  
 cell isolation  
 ex vivo study  
 cell lineage  
 phenotype  
 embryonal tissue  
 spinal cord injury: TH, therapy  
 diagnostic value  
 Alzheimer disease: DI, diagnosis  
 Parkinson disease: DI, diagnosis  
 neurologic disease: DI, diagnosis  
 human  
 nonhuman  
 review
- L17 ANSWER 46 OF 63 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN  
 AB Progressive neuronal loss in Alzheimer's disease (AD) is considered to be a consequence of the neurotoxic properties of amyloid- $\beta$  peptides (A $\beta$ ). T-817MA (1-{3-[2-(1-benzothiophen-5-yl) ethoxy] propyl}-3-azetidinol. . . . therapeutic agent for the treatment of AD based on its neuroprotective potency against A $\beta$ -induced neurotoxicity and its effect of enhancing axonal regeneration in the sciatic nerve axotomy model. The stress-induced neurotoxicity was assessed.  
 CT Medical Descriptors:  
 \*neurotoxicity  
 \*nerve injury  
 \*nerve fiber growth  
 \*neuroprotection  
 nerve cell  
 central nervous system  
 Alzheimer disease  
 drug potency  
 nerve fiber regeneration  
 nerve fiber transection  
 disease model  
 oxidative stress  
 coculture  
 brain cell  
 glia  
 nerve cell culture  
 hippocampus  
 degenerative disease  
 nerve cell necrosis  
 brain slice  
 treatment indication  
 nonhuman  
 female  
 rat  
 animal model  
 animal cell  
 article  
 priority journal
- \*1.  
 L17 ANSWER 47 OF 63 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN  
 AB . . . testosterone actions is neuroprotection. There are some evidences supporting the hypothesis that testosterone may act protectively in neurodegenerative disorders, e.g. Alzheimer's disease (AD), mild cognitive impairment (MCI) or depression. Androgens alter also the morphology, survival and axonal regeneration of motor neurons. These hormones accelerate the regeneration of hamster facial nerve and anterior tibialis sciatic nerve in rabbits following.  
 CT Medical Descriptors:  
 \*central nervous system disease: DT, drug therapy  
 \*central nervous system disease: PC, prevention  
 \*neuroprotection  
 Alzheimer disease: DT, drug therapy  
 Alzheimer disease: DT, etiology  
 Alzheimer disease: PC, prevention  
 cognitive defect: DT, drug therapy  
 cognitive defect: PC, prevention  
 depression: DT, drug therapy  
 depression: PC, prevention  
 nerve fiber regeneration  
 hormonal regulation  
 hamster  
 facial nerve  
 sciatic nerve  
 rabbit  
 nerve.
- L17 ANSWER 48 OF 63 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN  
 TI Modulation of axonal regeneration in neurodegenerative disease: Focus on Nogo.  
 AB Recent work has demonstrated that axonal regeneration in the central nervous system is limited by myelin-derived Nogo binding to an axonal Nogo Receptor. The Nogo system appears . . . a physiologic role in regulating structural plasticity. The possibility that the Nogo system contributes to pathologic and compensatory plasticity in Alzheimer's disease is considered.  
 CT Medical Descriptors:  
 \*nerve fiber regeneration  
 \*degenerative disease: DT, etiology  
 \*Alzheimer disease: DT, etiology  
 central nervous system  
 neuropathology  
 neurite  
 nerve cell plasticity  
 human  
 nonhuman  
 human cell  
 animal cell  
 article  
 protein: EC, endogenous compound  
 protein nogo: EC, endogenous compound  
 myelin: EC, endogenous compound  
 receptor: . . .
- L17 ANSWER 49 OF 63 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN  
 AB . . . estrogen in neuroprotection. Accumulated clinical evidence suggests that estrogen exposure decreases the risk and delays the onset and progression of Alzheimer's disease and schizophrenia, and may also enhance recovery from traumatic neurological injury such as

stroke. Recent basic science studies show. . . the classical nuclear estrogen receptor, through which estrogen alters expression of estrogen responsive genes that play a role in apoptosis, axonal regeneration, or general trophic support. Yet another possibility is that estrogen receptors in the membrane or cytoplasm alter phosphorylation cascades through.

Medical Descriptors:

\*neuroprotection  
\*schizophrenia: PC, prevention  
\*schizophrenia: DT, drug therapy  
\*stroke: PC, prevention  
\*stroke: DT, drug therapy  
\*Alzheimer disease: PC, prevention  
\*Alzheimer disease: DT, drug therapy

nervous system development  
brain injury: PC, prevention  
brain injury: DT, drug therapy  
genetic transcription

Glia

nerve cell

in vitro study

antioxidant activity

human

nonhuman

rat

animal.

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AB . . . Rho blocks the neurite growth-inhibitor effects of myelin-associated glycoprotein (MAG). These findings may have clinical applications in the stimulation of axonal regeneration following injury within the CNS, and possibly in the treatment of neurodegenerative disorders.

Medical Descriptors:

\*nerve fiber growth

cell structure

cell motility

degenerative disease: ET, etiology

nerve fiber regeneration

Alzheimer disease: ET, etiology

nonhuman

human cell

animal cell

short survey

\*rho factor: EC, endogenous compound

myelin associated glycoprotein: EC, endogenous compound

rho antagonist: DV, drug development

rho.

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AB Reactive oxygen species (ROS) are supposed to be involved in neurodegenerative processes like Parkinson's or Alzheimer's disease. Beside this there are an increasing number of studies indicating an involvement of ROS in traumatic brain injury. We . . . astrocytes are able to protect retinal ganglion cells against ROS-induced oxidative stress, (ii) astrocytes release soluble neurotrophic factors supporting RGC axonal regeneration, and (iii) free radical production after tissue injury may partly contribute to the failure of axonal regeneration in the adult mammalian central

nervous system.

L17 ANSWER 52 OF 63 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

TI Aberrant GAP-43 gene expression in Alzheimer's disease.  
AB GAP-43 is a growth-associated phosphoprotein expressed at high levels in neurons during development, axonal regeneration, and neuritic sprouting. . . GAP-43 gene expression in mature neurons is probably functionally important for the structural remodeling of synapses as required for learning and establishing new memory. The widespread aberrant neuritic growth accompanied by impaired synaptic plasticity in Alzheimer's disease (AD) suggests that abnormal GAP-43 gene expression may contribute to the cascade of neurodegeneration. In the present study, end-stage.

Medical Descriptors:

\*Alzheimer disease

article

brain cortex

chromosome aberration

gene expression

human

human tissue

lewy body

nerve cell plasticity

nerve degeneration

nerve fiber growth

priority journal

receptor down regulation

white matter

L17 ANSWER 53 OF 63 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

AB We have examined the possibility of promoting axonal regeneration within lesioned neural tissue using grafted artificial gel matrices. Polymeric matrices which feature a three-dimensional crosslinked macromolecular network were implanted. The deposition of newly synthesized extracellular molecules. This rearrangement of the brain scaffolding process into an organized cellular coating promoted axonal regeneration into the gels. Entrapment of embryonic neurons and embryonal carcinoma (EC)-derived neurons, within the gels, was performed to explore the.

Medical Descriptors:

\*neurosurgery

\*parkinson disease: SU, surgery

\*transplantation

Alzheimer disease: SU, surgery

animal experiment

animal tissue

controlled study

epilepsy: SU, surgery

human

human tissue

huntington chorea: SU, surgery

korsakoff psychosis: SU, surgery

nonhuman

short survey

L17 ANSWER 54 OF 63 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

AB . . . ganglia neurons was responsive to nerve growth factor (100 ng/ml). Nerve growth factor induced an increase of initial rate of axonal regeneration and influenced the survival time of these neurons. Acetyl-L-carnitine (250 µM) did not affect the axonal regeneration but substantially attenuated the

rate of neuronal mortality. A significant difference was evident between the acetyl-L-carnitine-treated and the untreated neurons.

CT

Medical Descriptors:  
\*aged  
\*nervous system  
\*spinal ganglion  
\*Alzheimer disease: ET, etiology  
animal cell  
article  
cell death  
cell protection  
cell survival  
controlled study  
drug effect  
male  
methodology  
nerve fiber degeneration  
neurotropism  
nonhuman  
rat  
\*levacarnine: PD, pharmacology  
\*nerve growth factor: PD, pharmacology  
\*neurotropic agent: PD.

L17 ANSWER 55 OF 63 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN  
AB . . . molecular techniques underlying nerve growth are discussed. Possible therapeutic approaches are presented for many neurologic disorders, ranging from stroke to Alzheimer's disease to acquired immunodeficiency syndrome, based on regrowing or saving injured neurons. The clinical neurologist will become important in practical applications and research into prolonging neuronal survival and fostering axonal regeneration. Over the coming years, with further research, it is anticipated that patients will be treated with these or similar modulatory.

L17 ANSWER 56 OF 63 TOXCENTER COPYRIGHT 2007 ACS on STN  
AB Progressive neuronal loss in Alzheimer's disease (AD) is considered to be a consequence of the neurotoxic properties of amyloid-beta peptides (A beta). T-817MA (1-[3-12-(1-benzothiophen-5-yl)]-agent for the treatment of AD based on its neuroprotective potency against A beta-induced neurotoxicity and its effect of enhancing axonal regeneration in the sciatic nerve axotomy model. The neuroprotective effect of T-817MA against A beta(1-42) or oxidative stress-induced neurotoxicity was assessed.

ST

of Organisms  
neuron: nervous system; central nervous system: nervous system; glial cell: nervous system; cortical neuron: nervous system

ST

Diseases  
Alzheimer's disease: nervous system disease, behavioral and mental disorders

ST

Chemicals & Biochemicals  
hydrogen peroxide; growth-associated protein 43; GSH; amyloid-beta: toxin.

L17 ANSWER 57 OF 63 TOXCENTER COPYRIGHT 2007 ACS on STN  
AB Progressive neuronal loss in Alzheimer's disease (AD) is considered to be a consequence of the neurotoxic properties of amyloid-beta peptides (A beta). T-817MA (1-[3-12-(1-benzothiophen-5-yl)]-ethoxy]. . . agent for the treatment of AD based on its neuroprotective potency against A beta-induced neurotoxicity and its effect of enhancing axonal regeneration in the sciatic nerve axotomy model. The neuroprotective effect of T-817MA against A beta(1-42) or oxidative

stress-induced neurotoxicity was assessed.

L17  
AB

ANSWER 58 OF 63 TOXCENTER COPYRIGHT 2007 ACS on STN  
Progressive neuronal loss in Alzheimer's disease (AD) is considered to be a consequence of the neurotoxic properties of amyloid-beta peptides (A beta). T-817MA was screened as a therapeutic agent for the treatment of AD based on its neuroprotective potency against A beta-induced neurotoxicity and its effect of enhancing axonal regeneration in the sciatic nerve axotomy model. The neuroprotective effect of T-817MA against A beta(1-42) or oxidative stress-induced neurotoxicity was assessed using.

L17  
AB

ANSWER 59 OF 63 TOXCENTER COPYRIGHT 2007 ACS on STN  
testosterone actions is neuroprotection. There are some evidences supporting the hypothesis that testosterone may act protectively in neurodegenerative disorders, e.g. Alzheimer's disease (AD), mild cognitive impairment (MCI) or depression. Androgens alter also the morphology, survival and axonal regeneration of motor neurons. These hormones accelerate the regeneration of hamster facial nerve and anterior tibialis sciatic nerve in rabbits following.

CT

Alzheimer Disease  
Depression  
Cognition Disorders

ST

nervous system; laryngeal motor nucleus: nervous system; motor neuron: nervous system; pelvic autonomic neuron: nervous system, spinal cord Diseases

ST

Alzheimer's disease: behavioral and mental disorders, nervous system disease, drug therapy  
Alzheimer Disease (MeSH)

ST

Diseases  
depression: behavioral and mental disorders, drug therapy  
Depression (MeSH)

ST

Diseases  
mild cognitive impairment: behavioral and mental.

L17  
AB

ANSWER 60 OF 63 TOXCENTER COPYRIGHT 2007 ACS on STN  
testosterone actions is neuroprotection. There is some evidence supporting the hypothesis that testosterone may act protectively in neurodegenerative disorders, e.g. Alzheimer's disease (AD) mild cognitive impairment (MCI), or depression. Androgens alter also the morphology, survival and axonal regeneration of motor neurons. These hormones accelerate the regeneration of hamster facial nerve and anterior tibialis sciatic nerve in rabbits following.

L17  
TI

ANSWER 61 OF 63 TOXCENTER COPYRIGHT 2007 ACS on STN  
Recent advance in adenoviral gene transfer technology for neuronal survival and axonal regeneration

AB

A review. Neuron-targeted gene transfer by adenovirus for the gene therapy of neuronal survival and axonal regeneration in the treatment of Parkinson's disease, Alzheimer's disease, malignant glioma etc. is reviewed.

L17  
AB

ANSWER 62 OF 63 TOXCENTER COPYRIGHT 2007 ACS on STN  
Reactive oxygen species (ROS) are supposed to be involved in neurodegenerative processes like Parkinson's or Alzheimer's disease. Beside this there are an increasing number of studies indicating an involvement of ROS in traumatic brain injury. We astrocytes are able to protect retinal ganglion cells against ROS-induced oxidative stress, (ii) astrocytes release soluble neurotrophic factors supporting axonal regeneration, and (iii) free radical production after tissue injury may partly contribute to the failure of axonal regeneration in the adult mammalian central nervous system.



L17 ANSWER 63 OF 63 TOXCENTER COPYRIGHT 2007 ACS ON STN  
 AB Reactive oxygen species (ROS) are supposed to be involved in neurodegenerative processes like Parkinson's or Alzheimer's disease. Beside this there are an increasing number of studies indicating an involvement of ROS in traumatic brain injury. We... astrocytes are able to protect retinal ganglion cells against ROS-induced oxidative stress, (ii) astrocytes release soluble neurotrophic factors supporting RGC axonal regeneration, and (iii) free radical production after tissue injury may partly contribute to the failure of axonal regeneration in the adult mammalian central nervous system.

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 (FILE 'HOME' ENTERED AT 15:48:48 ON 19 JUN 2007)  
 FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE, TOXCENTER' ENTERED AT 15:49:02 ON 19 JUN 2007  
 L1 12537 S ANYLOID(W)BETA(W)PEPTIDE  
 L2 9399 S BETA(W)ANYLOID(W)PEPTIDE  
 L3 13 S NOGO(W)RECEPTOR(W)ANTAGONIST  
 L4 0 S RETICULON(W)FAMILY(W)PEPTIDE  
 L5 459 S NOGO(W)RECEPTOR  
 L6 3 S L5 (P) (L1 OR L2)  
 L7 2 S NGRI(W)ANTAGONIST  
 L8 4 S L3 AND ALZHEIMER  
 L9 3 S L5 AND (L1 OR L2)  
 L10 10 S LINGO-1(W)ANTAGONIST  
 L11 0 S L10 AND (L1 OR L2)  
 L12 0 S L10 AND ALZHEIMER  
 L13 1 S L10 AND ALZHEIMER  
 L14 25 S NEP\*1-40\*  
 L15 0 S L14 AND (L1 OR L2)  
 L16 0 S L14 AND ALZHEIMER  
 L17 63 S ALZHEIMER AND (AXONAL (W)REGENERATION)  
 ==> s l17 and (l14 or l3 or l7)  
 L18 0 L17 AND (L14 OR L3 OR L7)  
 ==> s l17 and (l1 or l2)  
 L19 7 L17 AND (L1 OR L2)  
 ==> d l19 1-7  
 L19 ANSWER 1 OF 7 MEDLINE on STN  
 AN 2005316528 MEDLINE  
 DN PubMed ID: 15798005  
 TI A novel neurotrophic agent, T-817MA [1-{3-[2-(1-benzothiophen-5-yl)ethoxy]propyl}-3-azetidinol maleate], attenuates amyloid-beta-induced neurotoxicity and promotes neurite outgrowth in rat cultured central nervous system neurons.  
 AU Hirata Kazunari; Yamaguchi Hidetoshi; Takamura Yusaku; Takagi Akiko; Fukushima Tetsuo; Iwakami Noboru; Saitoh Akihito; Nakagawa Masaya; Yamada Tatsuo  
 CS Research Laboratories, Toyama Chemical Co., Ltd. 2-4-1 Shimookui, Toyama, 930-8508, Japan.; kazunari.hirata@toyama-chemical.co.jp  
 SO The Journal of pharmacology and experimental therapeutics, (2005 Jul) Vol. 314, No. 1, pp. 252-9. Electronic Publication: 2005-03-29.  
 Journal code: 0376362. ISSN: 0022-3565.  
 CY United States  
 DT (IN VITRO)  
 LA English

Priority Journals  
 200508  
 Entered STN: 21 Jun 2005  
 Last Updated on STN: 27 Aug 2005  
 Entered Medline: 26 Aug 2005  
 L19 ANSWER 2 OF 7 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN  
 AN 2005365391 BIOSIS  
 DN PREV200510151552  
 TI A novel neurotrophic agent, T-817MA [1-{3-[2-(1-benzothiophen-5-yl)ethoxy]propyl}-3-azetidinol maleate], attenuates amyloid-beta-induced neurotoxicity and promotes neurite outgrowth in rat cultured central nervous system neurons.  
 AU Hirata, Kazunari [Reprint Author]; Yamaguchi, Hidetoshi; Takamura, Yusaku; Takagi, Akiko; Fukushima, Tetsuo; Iwakami, Noboru; Saitoh, Akihito; Nakagawa, Masaya; Yamada, Tatsuo  
 CS Toyama Chem Co Ltd, Res Labs, 2-4-1 Shimookui, Toyama 9308508, Japan  
 SO Journal of Pharmacology and Experimental Therapeutics, (JUL 2005) Vol. 314, No. 1, pp. 252-259. http://www.jpvet.org.  
 CODEN: JPETAB. ISSN: 0022-3565.  
 DT Article  
 LA English  
 ED Entered STN: 14 Sep 2005  
 Last Updated on STN: 14 Sep 2005  
 L19 ANSWER 3 OF 7 CAPLUS COPYRIGHT 2007 ACS ON STN  
 AN 2005:603092 CAPLUS  
 DN 143:241806  
 TI A novel neurotrophic agent, T-817MA [1-{3-[2-(1-benzothiophen-5-yl)ethoxy]propyl}-3-azetidinol maleate], attenuates amyloid-beta-induced neurotoxicity and promotes neurite outgrowth in rat cultured central nervous system neurons  
 AU Hirata, Kazunari; Yamaguchi, Hidetoshi; Takamura, Yusaku; Takagi, Akiko; Fukushima, Tetsuo; Iwakami, Noboru; Saitoh, Akihito; Nakagawa, Masaya; Yamada, Tatsuo  
 CS Research Laboratories, Toyama Chemical Co., Ltd., Toyama, Japan  
 SO Journal of Pharmacology and Experimental Therapeutics (2005), 314(1), 252-259  
 CODEN: JPETAB, ISSN: 0022-3565  
 PB American Society for Pharmacology and Experimental Therapeutics  
 DT Journal  
 LA English  
 RE.CNT 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD  
 ALL CITATIONS AVAILABLE IN THE RE FORMAT  
 L19 ANSWER 4 OF 7 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN  
 AN 2005339732 EMBASE  
 TI A novel neurotrophic agent, T-817MA [1-{3-[2-(1-benzothiophen-5-yl)ethoxy]propyl}-3-azetidinol maleate], attenuates amyloid-beta-induced neurotoxicity and promotes neurite outgrowth in rat cultured central nervous system neurons.  
 AU Hirata K.; Yamaguchi H.; Takamura Y.; Takagi A.; Fukushima T.; Iwakami N.; Saitoh A.; Nakagawa M.; Yamada T.  
 CS K. Hirata, Research Laboratories, Toyama Chemical Co., Ltd., 2-4-1 Shimookui, Toyama, 930-8508, Japan. kazunari.hirata@toyama-chemical.co.jp  
 SO Journal of Pharmacology and Experimental Therapeutics, (2005) Vol. 314, No. 1, pp. 252-259.  
 Refs: 38  
 ISSN: 0022-3565 CODEN: JPETAB  
 CY United States  
 DT Journal; Article  
 FS 008 Neurology and Neurosurgery

030 Pharmacology  
037 Drug Literature Index  
LA English  
SL English  
ED Entered STN: 1 Sep 2005  
Last Updated on STN: 1 Sep 2005

ANSWER 5 OF 7 TOXCENTER COPYRIGHT 2007 ACS on STN  
AN 2005:250010 TOXCENTER  
CP Copyright (c) 2007 The Thomson Corporation  
DN PREV20051015152  
TI A novel neurotrophic agent, T-817WA [1-{3-[2-(1-benzothiophen-5-yl)ethoxy]propyl}-3-azetidinol maleate], attenuates amyloid-beta-induced neurotoxicity and promotes neurite outgrowth in rat cultured central nervous system neurons  
AU Hirata, Kazunari [Reprint Author]; Yamaguchi, Hidetoshi; Takamura, Yusaku; Takagi, Akiko; Fukushima, Tetsuo; Iwakami, Noboru; Saitoh, Akihito; Nakagawa, Masaya; Yamada, Tatsuo  
CS Toyama Chem Co Ltd, Res Labs, 2-4-1 Shimookui, Toyama 9308508, Japan  
SO Journal of Pharmacology and Experimental Therapeutics, (JUL 2005) Vol. 314, No. 1, pp. 252-259. http://www.jpet.org.  
CODEN: JPETAB. ISSN: 0022-3565.

DT Article  
FS BIOSIS  
OS BIOSIS 2005:365391  
LA English  
ED Entered STN: 20 Sep 2005  
Last Updated on STN: 20 Sep 2005

ANSWER 6 OF 7 TOXCENTER COPYRIGHT 2007 ACS on STN  
AN 2005:229107 TOXCENTER  
DN PubMed ID: 15798005  
TI A novel neurotrophic agent, T-817WA [1-{3-[2-(1-benzothiophen-5-yl)ethoxy]propyl}-3-azetidinol maleate], attenuates amyloid-beta-induced neurotoxicity and promotes neurite outgrowth in rat cultured central nervous system neurons  
AU Hirata Kazunari; Yamaguchi Hidetoshi; Takamura Yusaku; Takagi Akiko; Fukushima Tetsuo; Iwakami Noboru; Saitoh Akihito; Nakagawa Masaya; Yamada Tatsuo  
CS Research Laboratories, Toyama Chemical Co., Ltd, 2-4-1 Shimookui, Toyama, 930-8508, Japan. kazunari.hirata@toyama-chemical.co.jp  
SO The Journal of pharmacology and experimental therapeutics, (2005 Jul) Vol. 314, No. 1, pp. 252-9. Electronic Publication: 2005-03-29.  
Journal code: 0376362. ISSN: 0022-3565.

CY United States  
DT (IN VITRO)  
Journal; Article; (JOURNAL ARTICLE)  
MEDLINE 2005:14528  
OS MEDLINE  
LA English  
ED Entered STN: 30 Aug 2005  
Last Updated on STN: 30 Aug 2005

ANSWER 7 OF 7 TOXCENTER COPYRIGHT 2007 ACS on STN  
AN 2005:193359 TOXCENTER  
CP Copyright 2007 ACS  
DN CA143142418060  
TI A novel neurotrophic agent, T-817WA [1-{3-[2-(1-benzothiophen-5-yl)ethoxy]propyl}-3-azetidinol maleate], attenuates amyloid-beta-induced neurotoxicity and promotes neurite outgrowth in rat cultured central nervous system neurons  
AU Hirata, Kazunari; Yamaguchi, Hidetoshi; Takamura, Yusaku; Takagi, Akiko; Fukushima, Tetsuo; Iwakami, Noboru; Saitoh, Akihito; Nakagawa, Masaya;

CS Research Laboratories, Toyama Chemical Co., Ltd., Toyama, Japan.  
SO Journal of Pharmacology and Experimental Therapeutics, (2005) Vol. 314, No. 1, pp. 252-259.  
CODEN: JPETAB. ISSN: 0022-3565.

Yamada, Tatsuo  
Research Laboratories, Toyama Chemical Co., Ltd., Toyama, Japan.  
SO Journal of Pharmacology and Experimental Therapeutics, (2005) Vol. 314, No. 1, pp. 252-259.  
CODEN: JPETAB. ISSN: 0022-3565.

JAPAN  
CY Journal  
DT Journal  
OS CAPLUS 2005:603092  
LA English  
ED Entered STN: 19 Jul 2005  
Last Updated on STN: 29 Aug 2006

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ANSWER 1 OF 63 MEDLINE on STN  
L17 2007293203 IN-PROCESS  
DN PubMed ID: 17503736  
TI The pivotal role of RhoA GTPase in the molecular signaling of axon growth inhibition after CNS injury and targeted therapeutic strategies.  
AU Gross Robert E; Mei Qi; Gutekunst Claire-Anne; Torre Enrique  
CS Department of Neurosurgery, Center for Neurodegenerative Diseases, Emory University School of Medicine, Atlanta, GA 30322, USA.  
Robert.grossememoryhealthcare.org  
NC 5K08NS046322 (NINDS)  
SO Cell transplantation, (2007) Vol. 16, No. 3, pp. 245-62.  
Journal code: 9208854. ISSN: 0963-6897.

CY United States  
DT (RESEARCH SUPPORT, N.I.H., EXTRAMURAL)  
(RESEARCH SUPPORT, NON-U.S. GOV'T)  
English  
LA English  
FS NONMEDLINE; IN-PROCESS; NONINDEXED; Priority Journals  
ED Entered STN: 17 May 2007  
Last Updated on STN: 17 May 2007

ANSWER 2 OF 63 MEDLINE on STN  
AN 2007251491 IN-PROCESS  
DN PubMed ID: 17222456  
TI The role of chondroitin sulfate proteoglycans in regeneration and plasticity in the central nervous system.  
AU Galtrey Clare M; Fawcett James W  
CS Cambridge Centre for Brain Repair, Department of Clinical Neurosciences, University of Cambridge, Robinson Way, Cambridge, CB2 2PY, UK.  
Brain research reviews, (2007 Apr) Vol. 54, No. 1, pp. 1-18. Electronic Publication: 2007-01-11.  
Journal code: 101300366. ISSN: 0165-0173.

CY Netherlands  
DT Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, NON-U.S. GOV'T)  
English  
LA English  
FS NONMEDLINE; IN-PROCESS; NONINDEXED; Priority Journals  
ED Entered STN: 28 Apr 2007  
Last Updated on STN: 15 May 2007

ANSWER 3 OF 63 MEDLINE on STN  
AN 2007124185 MEDLINE  
DN PubMed ID: 17303085  
TI Two hydrophobic segments of the RTN1 family determine the ER localization and retention.  
AU Iwahashi Jun; Hamada Nobuyuki; Watanabe Hiroshi  
CS Division of Infectious Diseases, Department of Infectious Medicine, Kurume University School of Medicine, 67 Asahimachi, Kurume, Fukuoka 830-0011,

SO	Japan.. iwahashi@med.kurume-u.ac.jp Biochemical and biophysical research communications, (2007 Apr 6) Vol. 355, No. 2, pp. 508-12. Electronic Publication: 2007-02-07. Journal code: 0372516. ISSN: 0006-291X.
CY	United States Journal; Article; (JOURNAL ARTICLE)
DT	English Priority Journals
LA	200704
FS	Entered STN: 28 Feb 2007
EM	Last Updated on STN: 13 Apr 2007
ED	Entered Medline: 12 Apr 2007
L17	ANSWER 4 OF 63 MEDLINE on STN
AN	2006424547 IN-PROCESS
DN	PubMed ID: 16847745
Tl	Direct Rho-associated kinase inhibitor induces cofilin dephosphorylation and neurite outgrowth in PC-12 cells.
AU	Zhang Zhiqun; Ottens Andrew K; Larner Stephen F; Kobeissy Piras H; Williams Melissa L; Hayes Ronald L; Wang Kevin K W
CS	Centers for Neuroproteomics and Biomarkers Research, University of Florida, P.O. Box 100256, 100 S. Newell Drive, Gainesville, Florida, 32610, USA.
SO	Cellular & molecular biology letters, (2006) Vol. 11, No. 1, pp. 12-29. Journal code: 3607427. ISSN: 1425-8153.
CY	Poland Journal; Article; (JOURNAL ARTICLE)
DT	English
LA	NONMEDLINE; IN-DATA-REVIEW; IN-PROCESS; NONINDEXED; Priority Journals
FS	Entered STN: 19 Jul 2006
ED	Last Updated on STN: 12 Dec 2006
L17	ANSWER 5 OF 63 MEDLINE on STN
AN	2005314528 MEDLINE
DN	PubMed ID: 15798005
Tl	A novel neurotrophic agent, T-817MA [1-{3-[2-(1-benzothiophen-5-yl) ethoxy] propyl]-3-acetidinol maleate], attenuates amyloid-beta-induced neurotoxicity and promotes neurite outgrowth in rat cultured central nervous system neurons.
AU	Hirata Kazunari; Yamaguchi Hidetoshi; Takamura Yusaku; Takagi Akiko; Fukushima Tetsuo; Iwakami Noboru; Saitoh Akihito; Nakagawa Masaya; Yamada Tatsuo
CS	Research Laboratories, Toyama Chemical Co., Ltd., 2-4-1 Shimookui, Toyama, 930-8508, Japan.. kazunari.hirata@toyama-chemical.co.jp
SO	The Journal of pharmacology and experimental therapeutics, (2005 Jul) Vol. 314, No. 1, pp. 252-9. Electronic Publication: 2005-03-29. Journal code: 0376362. ISSN: 0022-3565.
CY	United States (IN VITRO)
DT	Journal; Article; (JOURNAL ARTICLE)
LA	English
FS	Priority Journals
EM	200508
ED	Entered STN: 21 Jun 2005 Last Updated on STN: 27 Aug 2005 Entered Medline: 26 Aug 2005
L17	ANSWER 6 OF 63 MEDLINE on STN
AN	2004619048 MEDLINE
DN	PubMed ID: 15591638
Tl	Neuroprotective role of testosterone in the nervous system.
AU	Bialek M; Zaremba P; Borowicz K K; Czuczwar S J
CS	Department of Pathophysiology, Skubiszewski Medical University, Jaczewskiego 8, PL 20-090 Lublin, Poland.
SO	Brain research, (1996 Dec 16) Vol. 743, No. 1-2, pp. 56-62. Journal code: 0045503. ISSN: 0006-8993.
SO	Polish journal of pharmacology, (2004 Sep-Oct) Vol. 56, No. 5, pp. 509-18 Ref: 87 Journal code: 931882. ISSN: 1230-6002.
CY	Poland Journal; Article; (JOURNAL ARTICLE)
DT	General Review; (REVIEW)
LA	English
FS	Priority Journals
EM	200507
ED	Entered STN: 20 Dec 2004 Last Updated on STN: 16 Jul 2005 Entered Medline: 15 Jul 2005
L17	ANSWER 7 OF 63 MEDLINE on STN
AN	2002451827 MEDLINE
DN	PubMed ID: 12212768
Tl	Modulation of axonal regeneration in neurodegenerative disease: focus on Nogo.
AU	Strittmatter Stephen M
CS	Department of Neurology, Yale University School of Medicine, New Haven, CT 06510, USA.. Stephen.Strittmatter@yale.edu
SO	Journal of molecular neuroscience : MN, (2002 Aug-Oct) Vol. 19, No. 1-2, pp. 117-21. Ref: 23 Journal code: 9002991. ISSN: 0895-8696.
CY	United States Journal; Article; (JOURNAL ARTICLE)
DT	(RESEARCH SUPPORT, NON-U.S. GOV'T) (RESEARCH SUPPORT, U.S. GOV'T, P.H.S.) General Review; (REVIEW)
LA	English
FS	Priority Journals
EM	200301
ED	Entered STN: 6 Sep 2002 Last Updated on STN: 29 Jan 2003 Entered Medline: 28 Jan 2003
L17	ANSWER 8 OF 63 MEDLINE on STN
AN	2001042902 MEDLINE
DN	PubMed ID: 11040417
Tl	Neuroprotection by estradiol.
AU	Garcia-Segura L M; Azcoitia I; DonCarlos L L
CS	Instituto Cajal, CSIC, Madrid, Spain
SO	Progress in neurobiology, (2001 Jan) Vol. 63, No. 1, pp. 29-60. Ref: 427 Journal code: 0370121. ISSN: 0301-0082.
CY	ENGLAND: United Kingdom Journal; Article; (JOURNAL ARTICLE)
DT	(RESEARCH SUPPORT, NON-U.S. GOV'T) General Review; (REVIEW)
LA	English
FS	Priority Journals
EM	200012
ED	Entered STN: 22 Mar 2001 Last Updated on STN: 22 Mar 2001 Entered Medline: 7 Dec 2000
L17	ANSWER 9 OF 63 MEDLINE on STN
AN	97169682 MEDLINE
DN	PubMed ID: 9017230
Tl	Postnatal retinal ganglion cells in vitro: protection against reactive oxygen species (ROS)-induced axonal degeneration by cocultured astrocytes.
AU	Lucius R; Sievers J
CS	Anatomisches Institut, Universitat Kiel, Germany.
SO	Brain research, (1996 Dec 16) Vol. 743, No. 1-2, pp. 56-62. Journal code: 0045503. ISSN: 0006-8993.

CY Netherlands  
 Journal; Article; (JOURNAL ARTICLE)  
 DT (RESEARCH SUPPORT, NON-U.S. GOV'T)  
 LA English  
 FS Priority Journals  
 EM 199704  
 ED Entered STN: 24 Apr 1997  
 Last Updated on STN: 24 Apr 1997  
 Entered Medline: 17 Apr 1997

L17 ANSWER 10 OF 63 MEDLINE on STN  
 AN 96010259 MEDLINE  
 DN PubMed ID: 7573369  
 TI Aberrant GAP-43 gene expression in Alzheimer's disease.  
 AU de la Monte S M; Ng S C; Hsu D W  
 CS Alzheimer's Disease Research Center, Neuropathology Laboratory, Massachusetts General Hospital, Harvard Medical School, Boston, USA.  
 NC R01-NS29793 (NINDS)  
 SO The American journal of pathology. (1995 Oct) Vol. 147, No. 4, pp. 934-46.  
 Journal code: 0370502. ISSN: 0002-9440.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Abridged Index Medicus Journals; Priority Journals  
 EM 199511  
 ED Entered STN: 27 Dec 1995  
 Last Updated on STN: 6 Feb 1998  
 Entered Medline: 9 Nov 1995

L17 ANSWER 11 OF 63 MEDLINE on STN  
 AN 90055926 MEDLINE  
 DN PubMed ID: 2573331  
 TI Growth factors for neuronal survival and process regeneration. Implications in the mammalian central nervous system.  
 AU Lipton S A  
 CS Department of Neurology, Children's Hospital, Boston, MA 02115.  
 NC EY05477 (NEI)  
 EY06087 (NEI)  
 NS00879 (NINDS)  
 SO Archives of neurology. (1989 Nov) Vol. 46, No. 11, pp. 1241-8. Ref: 109  
 Journal code: 0372436. ISSN: 0003-9942.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 (RESEARCH SUPPORT, NON-U.S. GOV'T)  
 (RESEARCH SUPPORT, U.S. GOV'T, NON-P.H.S.)  
 (RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)  
 General Review; (REVIEW)  
 LA English  
 FS Abridged Index Medicus Journals; Priority Journals  
 EM 198912  
 ED Entered STN: 28 Mar 1990  
 Last Updated on STN: 3 Feb 1997  
 Entered Medline: 12 Dec 1989

L17 ANSWER 12 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN  
 AN 2007-248532 BIOSIS  
 DN PREV200700244493  
 TI Two hydrophobic segments of the RTN1 family determine the ER localization and retention.  
 AU Iwahashi, Jun [Reprint Author]; Hamada, Nobuyuki; Watanabe, Hiroshi  
 Kurume Univ, Sch Med, Dept Infect Med, Div Infect Dis, 67 Asahimachi, Kurume, Fukuoka 8300011, Japan

iwahashi@med.kurume-u.ac.jp  
 Biochemical and Biophysical Research Communications, (APR 6 2007) Vol. 355, No. 2, pp. 508-512.  
 CODEN: BBRCA9. ISSN: 0006-291X.  
 DT Article  
 LA English  
 ED Entered STN: 18 Apr 2007  
 Last Updated on STN: 18 Apr 2007

L17 ANSWER 13 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN  
 AN 2006-539139 BIOSIS  
 DN PREV200600538678  
 TI Direct Rho-associated kinase inhibitor induces cofilin dephosphorylation and neurite outgrowth in PC-12 cells.  
 AU Zhang, Zhiquan; Ottens, Andrew K.; Larner, Stephen F.; Kobeissy, Firas H.; Williams, Melissa L.; Hayes, Ronald L.; Wang, Kevin K. W. [Reprint Author]  
 Univ Florida, Ctr Neuroprote and Biomarkers Res, McKnight Brain Inst, POB 100256, 100 S Newell Dr, Gainesville, FL 32610 USA  
 kwang@psychiatry.ufl.edu  
 SO Cellular & Molecular Biology Letters, (MAR 2006) Vol. 11, No. 1, pp. 12-29.  
 ISSN: 1425-8153.  
 DT Article  
 LA English  
 ED Entered STN: 18 Oct 2006  
 Last Updated on STN: 18 Oct 2006

L17 ANSWER 14 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN  
 AN 2005-365391 BIOSIS  
 DN PREV200510151552  
 TI A novel neurotrophic agent, T-817MA [1-{3-[2-(1-benzothiophen-5-yl)ethoxy]propyl}-3-azetidinol maleate], attenuates amyloid-beta-induced neurotoxicity and promotes neurite outgrowth in rat cultured central nervous system neurons.  
 AU Hirata, Kazunari [Reprint Author]; Yamaguchi, Hidetoshi; Takamura, Yusaku; Takagi, Akiko; Fukushima, Tetsuo; Iwakami, Noboru; Saitoh, Akihito; Nakagawa, Masaya; Yamada, Tatsuo  
 Toyama Chem Co Ltd, Res Labs, 2-4-1 Shimookui, Toyama 9308508, Japan  
 kazunari.hirata@toyama-chemical.co.jp  
 SO Journal of Pharmacology and Experimental Therapeutics, (JUL 2005) Vol. 314, No. 1, pp. 252-259. http://www.jpet.org.  
 CODEN: JPETAB. ISSN: 0022-3565.  
 DT Article  
 LA English  
 ED Entered STN: 14 Sep 2005  
 Last Updated on STN: 14 Sep 2005

L17 ANSWER 15 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN  
 AN 2005-211699 BIOSIS  
 DN PREV20050020216  
 TI Neuroprotective role of testosterone in the nervous system.  
 AU Bialek, Magdalena; Zaremba, Pawel; Borowicz, Kinga K.; Czuczwar, Stanislaw J. [Reprint Author]  
 Dept Pathophysiol, Skubiszewski Med Univ, Jaczewskiego 8, PL-20090, Lublin, Poland  
 czuczwar@eyahoo.com  
 SO Polish Journal of Pharmacology, (September 2004) Vol. 56, No. 5, pp. 509-518. Print.  
 ISSN: 1230-6002 (ISSN print).  
 DT Article  
 General Review; (Literature Review)

LA English  
ED Entered STN: 1 Jun 2005  
Last Updated on STN: 1 Jun 2005

L17 ANSWER 16 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN  
AN 2002:525476 BIOSIS  
DN PREV200200525476  
TI Modulation of axonal regeneration in neurodegenerative disease: Focus on Nogo.  
AU Strittmatter, Stephen M. [Reprint author]  
CS Department of Neurology, and Section of Neurobiology, Yale University School of Medicine, P.O. Box 208018, New Haven, CT, 06510, USA  
Stephen.Strittmatter@yale.edu  
SO Journal of Molecular Neuroscience, (August-October, 2002) Vol. 19, No. 1-2, pp. 117-121. print.  
CODEN: JMNES. ISSN: 0895-8696.

DT Article  
LA English  
ED Entered STN: 9 Oct 2002  
Last Updated on STN: 9 Oct 2002

L17 ANSWER 17 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN  
AN 2001:560175 BIOSIS  
DN PREV200100560175  
TI Neurofilament protein in cerebrospinal fluid: A marker of white matter changes.  
AU Sjogren, M. [Reprint author]; Blomberg, M.; Jonsson, M.; Wahlund, L.-O.; Edman, A.; Lind, K.; Rosengren, L.; Blennow, K.; Wallin, A.  
CS Institute of Clinical Neuroscience, Sahlgrenska University Hospital/Molndal, SE 431 80, Molndal, Sweden  
magnus.sjogren@medfak.gu.se  
SO Journal of Neuroscience Research, (November 1, 2001) Vol. 66, No. 3, pp. 510-516. print.  
CODEN: JNREDK. ISSN: 0360-4012.

DT Article  
LA English  
ED Entered STN: 5 Dec 2001  
Last Updated on STN: 25 Feb 2002

L17 ANSWER 18 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN  
AN 2000:148411 BIOSIS  
DN PREV200000148411  
TI Anti-amnesic effect of TEI-3356, a stable analogue of prostacyclin, assessed with amyloid beta(1-42) protein-infused rats.  
AU Suwa, Yotomasa [Reprint author]; Yamada, Kiyofumi; Atai, Takami [Reprint author]; Sakurai, Katsutoshi [Reprint author]; Yoshioka, Noboru [Reprint author]; Nabeshima, Toshitaka  
CS Institute for Biomedical Research, Teijin Ltd., Hino, Tokyo, 191-8512, Japan  
SO Society for Neuroscience Abstracts, (1999) Vol. 25, No. 1-2, pp. 2122. print.  
Meeting Info.: 29th Annual Meeting of the Society for Neuroscience. Miami Beach, Florida, USA. October 23-28, 1999. Society for Neuroscience. ISSN: 0190-5295.  
Conference, (Meeting)  
DT Article  
LA English  
ED Entered STN: 19 Apr 2000  
Last Updated on STN: 4 Jan 2002

L17 ANSWER 19 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

AN 1997:75938 BIOSIS  
DN PREV199799382641  
TI Postnatal retinal ganglion cells in vitro: Protection against reactive oxygen species (ROS)-induced axonal degeneration by cocultured astrocytes.  
AU Lucius, Ralph [Reprint author]; Sievers, Jobst  
CS Anatomisches Inst., Universitaet Kiel, Olshausenstr. 40, D-24118 Kiel, Germany  
SO Brain Research, (1996) Vol. 743, No. 1-2, pp. 56-62.  
CODEN: BRREP. ISSN: 0006-8993.

DT Article  
LA English  
ED Entered STN: 26 Feb 1997  
Last Updated on STN: 26 Feb 1997

L17 ANSWER 20 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN  
AN 1995:511111 BIOSIS  
DN PREV199598516161  
TI Aberrant GAP-43 gene expression in Alzheimer's disease.  
AU De La Monte, Suzanne M. [Reprint author]; Ng, Shi-Chung; Hsu, Dora W.  
CS Massachusetts Gen. Hosp., MGH Cancer Cent.-MGH East, 149 13th St., 7th Floor, Charlestown, MA 02129, USA  
SO American Journal of Pathology, (1995) Vol. 147, No. 4, pp. 934-946.  
CODEN: AJPA44. ISSN: 0002-9440.

DT Article  
LA English  
ED Entered STN: 29 Nov 1995  
Last Updated on STN: 29 Nov 1995

L17 ANSWER 21 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN  
AN 1993:391353 BIOSIS  
DN PREV199396066553  
TI Disrupted beta-1-adrenoceptor-G protein coupling in the temporal cortex of patients with Alzheimer's disease.  
AU Cowburn, Richard F. [Reprint author]; Vestling, Monika; Fowler, Christopher J.; Ravid, Rivka; Winblad, Bengt; O'Neill, Cora  
CS Dep. Geriatr. Med. B56, Huddinge Univ. Hosp., 141 86 Huddinge, Sweden  
SO Neuroscience Letters, (1993) Vol. 155, No. 2, pp. 163-166.  
CODEN: NELED5. ISSN: 0304-3940.

DT Article  
LA English  
ED Entered STN: 23 Aug 1993  
Last Updated on STN: 23 Aug 1993

L17 ANSWER 22 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN  
AN 1993:345493 BIOSIS  
DN PREV199396042493  
TI The carboxy-terminus of the beta amyloid protein is critical for the seeding of amyloid formation: Implications for the pathogenesis of Alzheimer's disease.  
AU Jarrett, Joseph T.; Berger, Elizabeth P.; Lansbury, Peter T., Jr. [Reprint author]  
CS Dep. Chem., Mass. Inst. Technol., Cambridge, MA 02139, USA  
SO Biochemistry, (1993) Vol. 32, No. 18, pp. 4693-4697.  
CODEN: BICHAM. ISSN: 0006-2960.

DT Article  
LA English  
ED Entered STN: 26 Jul 1993  
Last Updated on STN: 31 Aug 1993

L17 ANSWER 23 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

AN 1992:526818 BIOSIS  
 DN PREV199294114893; BA94-134893  
 TI AUTOMATED MEDIAL SEPTAL-DIAGONAL BAND NEURONS EXPRESS JUN-LIKE IMMUNOREACTIVITY.  
 AU DRAGUNOV M [Reprint author]  
 CS DEP PHARMACOLOGY, FACULTY MEDICINE, UNIVERSITY AUCKLAND, PRIVATE BAG, AUCKLAND, NEW ZEALAND  
 SO Molecular Brain Research, (1992) Vol. 15, No. 1-2, PP. 141-144.  
 CODEN: MBREEA. ISSN: 0169-328X.  
 DT Article  
 FS BA  
 LA ENGLISH  
 ED Entered STN: 19 Nov 1992  
 Last Updated on STN: 19 Nov 1992  
 L17 ANSWER 24 OF 63 CAPLUS COPYRIGHT 2007 ACS on STN  
 AN 2007:366937 CAPLUS  
 TI The role of chondroitin sulfate proteoglycans in regeneration and plasticity in the central nervous system  
 AU Galtrey, Clare M.; Fawcett, James W  
 CS Cambridge Centre for Brain Repair, Department of Clinical Neurosciences, University of Cambridge, Cambridge, CB2 2PY, UK  
 SO Brain Research Reviews (2007), 54(1), 1-18  
 CODEN: BRERD2; ISSN: 0165-0173  
 PB Elsevier B.V.  
 DT Journal  
 LA English  
 L17 ANSWER 25 OF 63 CAPLUS COPYRIGHT 2007 ACS on STN  
 AN 2007:220128 CAPLUS  
 DN 146:302160  
 TI Nogo receptor (Ngr) disulfide structure, Ngr signaling inhibiting Ngr fragments, mutants, fusion products and genetic constructs, and uses in mediating axonal growth  
 IN Wen, Dingyi; Lee, Daniel H. S.; Pepinsky, R. Blake  
 PA Biogen Idec Ma Inc., USA  
 SO PCT Int. Appl., 89pp.  
 CODEN: PIXXD2  
 DT Patent  
 LA English  
 FAN.CNT 1  
 PATENT NO. KIND DATE APPLICATION NO. DATE  
 PI WO 2007025219 A2 20070301 WO 2006-US33369 20060825  
 W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW  
 RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CL, CM, GA, GN, GO, GW, ML, MR, NE, SN, TD, TG, BM, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, EA, EP, OA  
 P 20050825  
 PRAI US 2005-710864P  
 L17 ANSWER 26 OF 63 CAPLUS COPYRIGHT 2007 ACS on STN  
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 TI Two hydrophobic segments of the RTN1 family determine the ER localization and retention

AU Iwahashi, Jun; Hamada, Nobuyuki; Watanabe, Hiroshi  
 CS Division of Infectious Diseases, Department of Infectious Medicine, Kurume University School of Medicine, Kurume, Fukuoka, 830-0011, Japan  
 SO Biochemical and Biophysical Research Communications (2007), 355(2), 508-512  
 CODEN: BBRC9; ISSN: 0006-291X  
 PB Elsevier  
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 LA English  
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 CS Centers for Neuroproteomics and Biomarkers Research and Traumatic Brain Injury Studies, Departments of Neuroscience, McKnight Brain Institute, University of Florida, Gainesville, FL, 32610, USA  
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 CODEN: CMBLFF; ISSN: 1425-8153  
 PB University of Wroclaw, Institute of Biochemistry, Dep. of Genetic Biochemistry  
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 PA Wyeth, John, and Brother Ltd., USA; King's College London  
 SO PCT Int. Appl., 60pp.  
 CODEN: PIXXD2  
 DT Patent  
 LA English  
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 RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CL, CM, GA, GN, GO, GW, ML, MR, NE, SN, TD, TG, BM, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM  
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RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, NG, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, MG, TJ, TM

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 CS Research Laboratories, Toyama Chemical Co., Ltd., Toyama, Japan  
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 CS Department of Neurology, and Section of Neurobiology, Yale University School of Medicine, New Haven, CT, 06510, USA  
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 CS Instituto Cajal, C.S.I.C., Madrid, E-28002, Spain  
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 CODEN: PGNBAS; ISSN: 0301-0082  
 PB Elsevier Science Ltd.  
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 CODEN: FIMXD2  
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 RM: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, CA, GN, GW, ML, MR, NE, SN, TD, TG  
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 CS Anatomisches Institut der Universitaet Kiel, Olshausenstr. 40, Kiel,  
 D-24118, Germany  
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 CODEN: BEREAP; ISSN: 0006-8993  
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 CS Harvard Medical School, Massachusetts General Hospital, Boston, MA, 02129,  
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 CS J.W. Fawcett, Cambridge Centre for Brain Repair, Department of Clinical  
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 United Kingdom. jf108@cam.ac.uk  
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 FS 021 Developmental Biology and Teratology  
 008 Neurology and Neurosurgery  
 LA English  
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ISSN: 0301-0082 CODEN: PGNBAS  
PUI S 0301-0082(00)00025-3  
CY United Kingdom  
DT Journal, General Review  
FS 020 Gerontology and Geriatrics  
022 Human Genetics  
029 Clinical Biochemistry  
030 Endocrinology  
033 Pharmacology  
037 Drug Literature Index  
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DT Journal, (Short Survey)

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ED Entered STN: 2 Dec 1999  
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ISSN: 0006-8993 CODEN: BRREAP  
PUI S 0006-8993(96)01029-3  
CY Netherlands  
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FS 002 Physiology  
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037 Drug Literature Index  
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DN PubMed ID: 15798005  
TI A novel neurotrophic agent, T-817MA [1-{3-[2-(1-benzothiophen-5-yl)ethoxy]propyl}-3-azetidinol maleate], attenuates amyloid-beta-induced neurotoxicity and promotes neurite outgrowth in rat cultured central nervous system neurons  
AU Hirata Kazunari; Yamaguchi Hidetoshi; Takamura Yusaku; Takagi Akiko; Fukushima Tetsuo; Iwakami Noboru; Saitoh Akihito; Nakagawa Masaya; Yamada Tatsuo  
CS Research Laboratories, Toyama Chemical Co., Ltd, 2-4-1 Shimookui, Toyama, 930-8508, Japan. kazunari.hirata@toyama-chemical.co.jp  
SO The Journal of Pharmacology and Experimental Therapeutics, (2005 Jul) Vol. 314, No. 1, pp. 252-9. Electronic Publication: 2005-03-29.  
Journal code: 0376362. ISSN: 0022-3565.  
CY United States  
DT (IN VITRO)  
FS MEDLINE  
OS MEDLINE 2005:316528  
\*LA English  
ED Entered STN: 30 Aug 2005  
Last Updated on STN: 30 Aug 2005

=> d his

(FILE 'HOME' ENTERED AT 15:48:48 ON 19 JUN 2007)

FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE, TOXCENTER' ENTERED AT 15:49:02 ON 19 JUN 2007

L1 12537 S AMYLOID(W)BETA(W)PEPTIDE  
L2 9399 S BETA(W)AMYLOID(W)PEPTIDE  
L3 13 S NOGO(W)RECEPTOR(W)ANTAGONIST  
L4 0 S RETICULON(W)FAMILY(W)PEPTIDE  
L5 459 S NOGO(W)RECEPTOR  
L6 3 S L5 (P) (L1 OR L2)  
L7 2 S NGRI(W)ANTAGONIST  
L8 4 S L3 AND ALZHEIMER  
L9 3 S L5 AND (L1 OR L2)  
L10 10 S LINGO-1(W)ANTAGONIST  
L11 0 S L10 AND (L1 OR L2)  
L12 0 S L10 AND ALZHEIMER  
L13 1 S L10 AND ALZHEIMER  
L14 25 S NEP\*1-40\*  
L15 0 S L14 AND (L1 OR L2)  
L16 0 S L14 AND ALZHEIMER  
L17 63 S ALZHEIMER AND (AXONAL (W)REGENERATION)  
L18 0 S L17 AND (L14 OR L3 OR L7)  
L19 7 S L17 AND (L1 OR L2)  
=>

L17 ANSWER 54 OF 63 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN  
AN 92286708 EMBASE  
DN 1992286708  
TI Culture of dorsal root ganglion neurons from aged rats: Effects of acetyl-L-carnitine and NGF  
AU Manfredi A.; Forloni G.L.; Arrigoni-Martelli E.; Mancina M.  
CS Istituto di Fisiologia Umana II, Università degli Studi di Milano, Via Mangagalli 32, 20133 Milano, Italy  
SO International Journal of Developmental Neuroscience, (1992) Vol. 10, No. 4, pp. 321-329.  
ISSN: 0736-5748 CODEN: IJDNDE  
CY United Kingdom  
DT Journal; Article  
FS 001 Anatomy, Anthropology, Embryology and Histology  
008 Neurology and Neurosurgery  
020 Gerontology and Geriatrics  
030 Pharmacology  
037 Drug Literature Index  
LA English  
SL English  
ED Entered STN: 25 Oct 1992  
Last Updated on STN: 25 Oct 1992

L17 ANSWER 55 OF 63 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN  
AN 8268444 EMBASE  
DN 198268444  
TI Growth factors for neuronal survival and process regeneration. Implications in the mammalian central nervous system.  
AU Lipton S.A.  
CS Children's Hospital, Boston, MA 02115, United States  
SO Archives of Neurology, (1989) Vol. 46, No. 11, pp. 1241-1248.  
ISSN: 0003-9942 CODEN: ARNEAS  
CY United States  
DT Journal  
FS 001 Anatomy, Anthropology, Embryology and Histology  
002 Physiology  
005 General Pathology and Pathological Anatomy  
008 Neurology and Neurosurgery  
LA English  
SL English  
ED Entered STN: 12 Dec 1991  
Last Updated on STN: 12 Dec 1991

L17 ANSWER 56 OF 63 TOXCENTER COPYRIGHT 2007 ACS on STN  
AN 2005:250010 TOXCENTER  
CP Copyright (c) 2007 The Thomson Corporation  
DN PREV200510151552  
TI A novel neurotrophic agent, T-817MA [1-{3-[2-(1-benzothiophen-5-yl)ethoxy]propyl}-3-azetidinol maleate], attenuates amyloid-beta-induced neurotoxicity and promotes neurite outgrowth in rat cultured central nervous system neurons  
AU Hirata, Kazunari [Reprint Author]; Yamaguchi, Hidetoshi; Takamura, Yusaku; Takagi, Akiko; Fukushima, Tetsuo; Iwakami, Noboru; Saitoh, Akihito; Nakagawa, Masaya; Yamada, Tatsuo  
CS Toyama Chem Co Ltd, Res Labs, 2-4-1 Shimookui, Toyama 9308508, Japan  
kazunari.hirata@toyama-chemical.co.jp  
SO Journal of Pharmacology and Experimental Therapeutics, (JUL 2005) Vol.

=> s 11 and 12 and function  
L20 79 L1 AND L2 AND FUNCTION

=> s 11 or 12 (p) function  
L21 13364 L1 OR L2 (P) FUNCTION

=> s (11 or 12) and function  
L22 3236 (L1 OR L2) AND FUNCTION

=> s 122 and (13 or 17 or 114)  
MISSING OPERATOR L3 OR L7  
The search profile that was entered contains terms or nested terms that are not separated by a logical operator.

=> s 122 and (13 or 17 or 114)  
L23 0 L22 AND (L3 OR L7 OR L14)

=> d 120 70-79

L20 ANSWER 70 OF 79 TOXCENTER COPYRIGHT 2007 ACS on STN  
AN 2003:73424 TOXCENTER  
CP Copyright (c) 2007 The Thomson Corporation  
DN PREV20030015393  
TI Activation of Wnt signaling rescues neurodegeneration and behavioral  
impairments induced by beta-amyloid fibrils  
De Ferrari, G. V.; Chacon, M. A.; Barria, M. I.; Garrido, J. L.; Godoy, J.  
A.; Olivares, G.; Reyes, A. E.; Alvarez, A.; Bronfman, M.; Inestrosa, N.  
C. [Reprint Author]  
CS Molecular Neurobiology Unit, P Catholic University of Chile, PO Box 114-D,  
Santiago, Chile minestr@genes.bio.puc.cl  
SO Molecular Psychiatry, (2003) Vol. 8, No. 2, pp. 195-208. print.  
ISSN: 1359-4184.  
DT Article  
FS BIOSIS  
OS BIOSIS 2003:153993  
LA English  
ED Entered STN: 1 Apr 2003  
Last Updated on STN: 1 Apr 2003

L20 ANSWER 71 OF 79 TOXCENTER COPYRIGHT 2007 ACS on STN  
AN 2002:136467 TOXCENTER  
CP Copyright 2007 ACS  
DN CAL3626396235M  
TI The protective effects of melatonin from oxidative damage induced by  
amyloid beta-peptide 25-35 in middle-aged rats  
Shen, Y. X.; Xu, S. Y.; Wei, W.; Sun, X. X.; Liu, L. H.; Yang, J.; Dong,  
C.  
CS Institute of Clinical Pharmacology, Anhui Medical University, Hefei,  
230032, Peop. Rep. China.  
SO Journal of Pineal Research, (2002) Vol. 32, No. 2, pp. 85-89.  
CODEN: JPRSES. ISSN: 0742-3098.  
CY CHINA  
DT Journal  
FS CAPLUS  
OS CAPLUS 2002:267661  
LA English  
ED Entered STN: 18 Jun 2002  
Last Updated on STN: 2 May 2006

L20 ANSWER 72 OF 79 TOXCENTER COPYRIGHT 2007 ACS on STN  
AN 2002:78163 TOXCENTER  
CP Copyright 2007 ACS  
DN CAL3711153285U

TI Suppressed expression of nicotinic acetylcholine receptors by nanomolar  
beta-amyloid peptides in PC12 cells  
Guan, Z.-Z.; Miao, H.; Tian, J.-Y.; Unger, C.; Nordberg, A.; Zhang, X.  
CS Division of Molecular Neuropharmacology, Department of Clinical, Huddinge  
University Hospital, Huddinge, Sweden.  
SO Journal of Neural Transmission, (2001) Vol. 108, No. 12, pp. 1417-1433.  
CODEN: JNTRF3. ISSN: 1435-1463.  
CY SWEDEN  
DT Journal  
FS CAPLUS  
OS CAPLUS 2002:228581  
LA English  
ED Entered STN: 3 Apr 2002  
Last Updated on STN: 29 Aug 2006

L20 ANSWER 73 OF 79 TOXCENTER COPYRIGHT 2007 ACS on STN  
AN 2001:213180 TOXCENTER  
CP Copyright 2007 ACS  
DN CAL3524339064K  
TI Amyloid beta peptide 1-40 and the  
function of rat hippocampal hemicholinium-3 sensitive choline  
carriers: effects of a proteolytic degradation in vitro  
Kristofikova, Zdena; Tejkalova, Hana; Kiaschka, Jan  
CS Prague Psychiatric Centre, Prague, 18103/8, Czech Rep..  
SO Neurochemical Research, (2001) Vol. 26, No. 3, pp. 203-212.  
CODEN: NEREDZ. ISSN: 0364-3190.  
CY CZECH REPUBLIC  
DT Journal  
FS CAPLUS  
OS CAPLUS 2001:557510  
LA English  
ED Entered STN: 27 Nov 2001  
Last Updated on STN: 26 Mar 2002

L20 ANSWER 74 OF 79 TOXCENTER COPYRIGHT 2007 ACS on STN  
AN 2001:5964 TOXCENTER  
CP Copyright 2000 Wiley-Liss, Inc.  
DN PubMed ID: 10956426  
TI Hyperzine A and tacrine attenuate beta-amyloid  
peptide-induced oxidative injury  
Xiao X Q; Wang R; Tang X C  
CS State Key Laboratory of Drug Research, Shanghai Institute of Materia  
Medica, Chinese Academy of Sciences, Shanghai, China  
SO Journal of neuroscience research, (2000 Sep 1) Vol. 61, No. 5, pp. 564-9.  
Journal code: 7600111. ISSN: 0360-4012.  
CY United States  
DT (COMPARATIVE STUDY)  
Journal, Article, (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, NON-U.S. GOV'T)  
FS MEDLINE  
OS MEDLINE 2001032559  
LA English  
ED Entered STN: 16 Nov 2001  
Last Updated on STN: 16 Nov 2001

L20 ANSWER 75 OF 79 TOXCENTER COPYRIGHT 2007 ACS on STN  
AN 2000:192645 TOXCENTER  
CP Copyright 2007 ACS  
DN CAL3324329417X  
TI Hyperzine A and tacrine attenuate beta-amyloid  
peptide-induced oxidative injury  
Xiao, Xiao Qiu; Wang, Rui; Tang, Xi Can  
CS State Key Laboratory of Drug Research, Shanghai Institute of Materia  
Medica, Chinese Academy of Sciences, Shanghai, 200031, Peop. Rep. China.

SO Journal of Neuroscience Research, (2000) Vol. 61, No. 5, pp. 564-569.  
 CODEN: JNREDA. ISSN: 0360-4012.  
 CY CHINA  
 DT Journal  
 FS CAPLUS  
 OS CAPLUS 2000:655982  
 LA English  
 ED Entered STN: 16 Nov 2001  
 Last Updated on STN: 21 Dec 2004

L20 ANSWER 76 OF 79 TOXCENTER COPYRIGHT 2007 ACS on STN  
 AN 2000:180205 TOXCENTER  
 CP Copyright 2007 ACS  
 DN CA1340809483T  
 TI  $\beta$ -Amyloid augments platelet aggregation. Reduced activity of familial  
 angiotensin-converting enzyme mutants  
 AU Wolozin, B.; Maheshwari, S.; Jones, C.; Dukoff, R.; Wallace, W.; Racchi,  
 M.; Nagula, S.; Shulman, N. R.; Sunderland, T.; Bush, A.  
 CS Section on Geriatric Psychiatry, NIMH, Bethesda, MD, 20892, USA.  
 SO Molecular Psychiatry, (1998) Vol. 3, No. 6, pp. 500-507.  
 CODEN: MOFSFO. ISSN: 1359-4184.  
 CY UNITED STATES  
 DT Journal  
 FS CAPLUS  
 OS CAPLUS 2000:569427  
 LA English  
 ED Entered STN: 16 Nov 2001  
 Last Updated on STN: 5 Mar 2002

L20 ANSWER 77 OF 79 TOXCENTER COPYRIGHT 2007 ACS on STN  
 AN 2000:124293 TOXCENTER  
 CP Copyright 2007 ACS  
 DN CA13221277675N  
 TI  $\beta$ -Amyloid-42 binds to  $\alpha 7$  nicotinic acetylcholine receptor with  
 high affinity: implications for Alzheimer's disease pathology  
 AU Wang, Houan-Yan; Lee, Daniel H. S.; D'Andrea, Michael R.; Peterson, Per A.;  
 Shank, Richard P.; Reitz, Allen B.  
 CS R. W. Johnson Pharmaceutical Research Institute, Spring House, PA,  
 19477-0776, USA.  
 SO Journal of Biological Chemistry, (2000) Vol. 275, No. 8, pp. 5626-5632.  
 CODEN: JBCHA3. ISSN: 0021-9258.  
 CY UNITED STATES  
 DT Journal  
 FS CAPLUS  
 OS CAPLUS 2000:156752  
 LA English  
 ED Entered STN: 16 Nov 2001  
 Last Updated on STN: 16 Apr 2002

L20 ANSWER 78 OF 79 TOXCENTER COPYRIGHT 2007 ACS on STN  
 AN 1997:217243 TOXCENTER  
 CP Copyright 2007 ACS  
 DN CA12807071065X  
 TI Na<sup>+</sup>/K<sup>+</sup>-ATPase activity, glucose transport, and glutamate transport induced  
 by amyloid  $\beta$ -peptide and iron  
 AU Keller, Jeffrey N.; Germeyer, Ariane; Begley, James G.; Mattson, Mark P.  
 CS Sanders-Brown Research Center on Aging, University of Kentucky, Lexington,  
 KY, USA.  
 SO Journal of Neuroscience Research, (1997) Vol. 50, No. 4, pp. 522-530.  
 CODEN: JNREDA. ISSN: 0360-4012.  
 CY UNITED STATES  
 DT Journal  
 FS CAPLUS

OS CAPLUS 1997:774254  
 LA English  
 ED Entered STN: 16 Nov 2001  
 Last Updated on STN: 5 Jun 2002

L20 ANSWER 79 OF 79 TOXCENTER COPYRIGHT 2007 ACS on STN  
 AN 1997:206759 TOXCENTER  
 CP Copyright 2007 ACS  
 DN CA127243298480  
 TI  $\beta$ -Amyloid-induced cerebrovascular endothelial dysfunction  
 AU Thomas, Tom; McLendon, Chris; Sutton, E. Truitt; Thomas, George  
 CS Departments of Psychiatry and Physiology, College of Medicine, Univ. South  
 Florida, Tampa, FL, 33613, USA.  
 SO Annals of the New York Academy of Sciences, (1997) Vol. 826, No.  
 Cerebrovascular Pathology in Alzheimer's Disease, pp. 447-451.  
 CODEN: ANYA99. ISSN: 0077-8923.  
 CY UNITED STATES  
 DT Journal  
 FS CAPLUS  
 OS CAPLUS 1997:700729  
 LA English  
 ED Entered STN: 16 Nov 2001  
 Last Updated on STN: 18 Jun 2002

=> d 120 1-69

L20 ANSWER 1 OF 79 MEDLINE on STN  
 AN 2006023704 MEDLINE  
 DN PubMed ID: 16303255  
 TI Transgenic mice over-expressing human beta-amyloid have functional  
 nicotinic alpha 7 receptors.  
 AU Spencer J P; Weil A; Hill K; Hussain I; Richardson J C; Cusdin F S; Chen Y  
 H; Randall A D  
 CS Neurology and GI CDD, GlaxoSmithKline, Harlow, Essex CM19 5AW, UK..  
 SO Neuroscience, (2006 Feb) Vol. 137, No. 3, pp. 795-805. Electronic  
 Publication: 2005-11-21  
 Journal code: 7605074. ISSN: 0306-4522.  
 CY United States  
 DT (IN VITRO)  
 LA English  
 FS Priority Journals  
 EM 200604  
 ED Entered STN: 14 Jan 2006  
 Last Updated on STN: 13 Apr 2006  
 Entered Medline: 12 Apr 2006

L20 ANSWER 2 OF 79 MEDLINE on STN  
 AN 2002690364 MEDLINE  
 DN PubMed ID: 12450488  
 TI Alzheimer's disease and the basal forebrain cholinergic system: relations  
 to  $\beta$ -amyloid peptides, cognition, and  
 treatment strategies  
 AU Auld Daniel S; Kornecook Tom J; Bastianetto Stephanie; Quirion Remi  
 CS Douglas Hospital Research Centre, 6875 Blvd Lasalle, Verdun, Que, Canada  
 H4H 1R3.  
 SO Progress in neurobiology, (2002 Oct) Vol. 68, No. 3, pp. 209-45. Ref: 504  
 Journal code: 0370121. ISSN: 0301-0082.  
 CY England: United Kingdom  
 DT Journal: Article; (JOURNAL ARTICLE)  
 (RESEARCH SUPPORT, NON-U.S. GOV'T)  
 General Review; (REVIEW)

LA English  
FS Priority Journals  
EM 200303  
ED Entered STN: 14 Dec 2002  
Last Updated on STN: 6 Mar 2003  
Entered Medline: 5 Mar 2003

L20 ANSWER 3 OF 79 MEDLINE on STN  
AN 2002327989 MEDLINE  
DN PubMed ID: 12071472  
TI The protective effects of melatonin from oxidative damage induced by amyloid beta-peptide 25-35 in middle-aged rats.  
AU Shen Y X; Xu S Y; Wei W; Sun X X; Liu L H; Yang J; Dong C  
CS Institute of Clinical Pharmacology, Anhui Medical University, Hefei, China. shenyx@mail.hf.ah.cn  
SO Journal of pineal research, (2002 Mar) Vol. 32, No. 2, pp. 85-9.  
Journal code: 8504412. ISSN: 0742-3098.  
CY Denmark  
DT Journal; Article; (JOURNAL ARTICLE)  
LA English  
FS Priority Journals  
EM 200211  
ED Entered STN: 20 Jun 2002  
Last Updated on STN: 11 Dec 2002  
Entered Medline: 8 Nov 2002

L20 ANSWER 4 OF 79 MEDLINE on STN  
AN 2002327706 MEDLINE  
DN PubMed ID: 12070316  
TI Charge states rather than propensity for beta-structure determine enhanced fibrillogenesis in wild-type Alzheimer's beta-amyloid peptide compared to E22Q Dutch mutant.  
AU Massi Francesca; Klimov D; Thirumalai D; Straub John E  
CS Department of Chemistry, Boston University, 590 Commonwealth Avenue, Boston, MA 02215, USA.  
NC R01 NS 41356-01 (NINDS)  
SO Protein science : a publication of the Protein Society, (2002 Jul) Vol. 11, No. 7, pp. 1639-47.  
Journal code: 9211750. ISSN: 0961-8368.  
CY United States  
DT Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, NON-U.S. GOV'T)  
(RESEARCH SUPPORT, U.S. GOV'T, NON-P.H.S.)  
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)  
LA English  
FS Priority Journals  
EM 200308  
ED Entered STN: 19 Jun 2002  
Last Updated on STN: 11 Dec 2002  
Entered Medline: 19 Aug 2003

L20 ANSWER 5 OF 79 MEDLINE on STN  
AN 2001126187 MEDLINE  
DN PubMed ID: 11137881  
TI Beta-amyloid (1-42) affects MTT reduction in astrocytes: implications for vesicular trafficking and cell functionality.  
AU Kerokoski P; Soinen H; Pirttilä T  
CS Department of Neuroscience and Neurology, University of Kupio, Finland. petri.kerokoski@uku.fi  
SO Neurochemistry international, (2001 Feb) Vol. 38, No. 2, pp. 127-34.  
Journal code: 8006959. ISSN: 0197-0186.  
CY England; United Kingdom  
DT Journal; Article; (JOURNAL ARTICLE)

(RESEARCH SUPPORT, NON-U.S. GOV'T)  
LA English  
FS Priority Journals  
EM 200102  
ED Entered STN: 22 Mar 2001  
Last Updated on STN: 22 Mar 2001  
Entered Medline: 22 Feb 2001

L20 ANSWER 6 OF 79 MEDLINE on STN  
AN 2001101045 MEDLINE  
DN PubMed ID: 11119646  
TI Energy landscape theory for Alzheimer's amyloid beta-peptide fibril elongation.  
AU Massi F; Straub J E  
CS Department of Chemistry, Boston University, Boston, Massachusetts 02115, USA.  
SO Proteins, (2001 Feb 1) Vol. 42, No. 2, pp. 217-29.  
Journal code: 8700181. ISSN: 0887-3585.  
CY United States  
DT (COMPARATIVE STUDY)  
Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, NON-U.S. GOV'T)  
(RESEARCH SUPPORT, U.S. GOV'T, NON-P.H.S.)  
LA English  
FS Priority Journals  
EM 200102  
ED Entered STN: 22 Mar 2001  
Last Updated on STN: 22 Mar 2001  
Entered Medline: 1 Feb 2001

L20 ANSWER 7 OF 79 MEDLINE on STN  
AN 2001032559 MEDLINE  
DN PubMed ID: 10956426  
TI Hyperkine A and tacrine attenuate beta-amyloid peptide-induced oxidative injury.  
AU Xiao X Q; Wang R; Tang X C  
CS State Key Laboratory of Drug Research, Shanghai Institute of Materia Medica, Chinese Academy of Sciences, Shanghai, China.  
SO Journal of neuroscience research, (2000 Sep 1) Vol. 61, No. 5, pp. 564-9.  
Journal code: 7600111. ISSN: 0360-4012.  
CY United States  
DT (COMPARATIVE STUDY)  
Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, NON-U.S. GOV'T)  
(RESEARCH SUPPORT, U.S. GOV'T, NON-P.H.S.)  
LA English  
FS Priority Journals  
EM 200011  
ED Entered STN: 22 Mar 2001  
Last Updated on STN: 22 Mar 2001  
Entered Medline: 30 Nov 2000

L20 ANSWER 8 OF 79 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN  
AN 2007:315288 BIOSIS  
DN PREV200700315208  
TI Abeta Peptide and Alzheimer's Disease: Celebrating a Century of Research.  
AU Barrow, CJ [Editor]; Small, DH [Editor]  
CS Ocean Nutr Canada, Dartmouth, NS, Canada  
SO Barrow, CJ [Editor]; Small, DH [Editor]. (2007) Abeta Peptide and Alzheimer's Disease: Celebrating a Century of Research.  
Publisher: SPRINGER, 233 SPRING STREET, NEW YORK, NY 10013, UNITED STATES.  
ISBN: 978-1-85233-961-6(H).  
DT Book  
LA English  
ED Entered STN: 24 May 2007

Last Updated on STN: 24 May 2007

L20 ANSWER 9 OF 79 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN  
 AN 2006:452694 BIOSIS  
 DN PREV200600251494  
 TI Transgenic mice over-expressing human beta-amyloid have functional  
 nicotinic alpha 7 receptors.  
 AU Spencer, J. P. [Reprint Author]; Weil, A.; Hill, K.; Hussain, I.;  
 Richardson, J. C.; Cusdin, F. S.; Chen, Y. H.; Randall, A. D.  
 CS GlaxoSmithKline Inc. Neurol and GI CEDD, Harlow CM19 5AW, Essex, UK  
 jon.p.spencer@sk.com  
 SO Neuroscience, (2006) Vol. 137, No. 3, pp. 795-805.  
 CODEN: NRSCDN. ISSN: 0306-4522.  
 DT Article  
 LA English  
 ED Entered STN: 26 Apr 2006  
 Last Updated on STN: 26 Apr 2006

L20 ANSWER 10 OF 79 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on  
 STN  
 AN 2003:390477 BIOSIS  
 DN PREV200300390477  
 TI beta-Amyloid regulation of presynaptic nicotinic receptors in rat  
 hippocampus and neocortex.  
 AU Dougherty, John J.; Wu, Jianlin; Nichols, Robert A. [Reprint Author]  
 CS Department of Pharmacology and Physiology, College of Medicine, Drexel  
 University (formerly MCP Hahnemann University), 245 North 15th Street,  
 Philadelphia, PA, 19102, USA  
 robert.nichols@drexel.edu  
 SO Journal of Neuroscience, (July 30 2003) Vol. 23, No. 17, pp. 6740-6747.  
 print.  
 ISSN: 0270-6474 (ISSN print).  
 DT Article  
 LA English  
 ED Entered STN: 27 Aug 2003  
 Last Updated on STN: 27 Aug 2003

L20 ANSWER 11 OF 79 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on  
 STN  
 AN 2003:153993 BIOSIS  
 DN PREV200300153993  
 TI Activation of Wnt signaling rescues neurodegeneration and behavioral  
 impairments induced by beta-amyloid fibrils.  
 AU De Ferrari, G. V.; Chacon, M. A.; Barria, M. I.; Garrido, J. L.; Godoy, J.  
 A.; Olivares, G.; Reyes, A. E.; Alvarez, A.; Bronfman, M.; Inestrosa, N.  
 C. [Reprint Author]  
 CS Molecular Neurobiology Unit, P Catholic University of Chile, PO Box 114-D,  
 Santiago, Chile  
 ninestrosa@bio.puc.cl  
 SO Molecular Psychiatry, (2003) Vol. 8, No. 2, pp. 195-208. print.  
 ISSN: 1359-4184 (ISSN print).  
 DT Article  
 LA English  
 ED Entered STN: 26 Mar 2003  
 Last Updated on STN: 26 Mar 2003

L20 ANSWER 12 OF 79 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on  
 STN  
 AN 2002:423365 BIOSIS  
 DN PREV200200423365  
 TI Charge states rather than propensity for beta-structure determine enhanced  
 fibrillogenesis in wild-type Alzheimer's beta-amyloid  
 peptide compared to E22Q Dutch mutant.  
 AU Massi, Francesca; Klimov, D.; Thirumalai, D.; Straub, John E. [Reprint

author]  
 CS Department of Chemistry, Boston University, 590 Commonwealth Avenue,  
 Boston, MA, 02215, USA  
 straub@bu.edu  
 SO Protein Science, (July, 2002) Vol. 11, No. 7, pp. 1639-1647. print.  
 ISSN: 0961-8368.  
 DT Article  
 LA English  
 ED Entered STN: 7 Aug 2002  
 Last Updated on STN: 7 Aug 2002

L20 ANSWER 13 OF 79 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on  
 STN  
 AN 2002:260830 BIOSIS  
 DN PREV200200260830  
 TI The protective effects of melatonin from oxidative damage induced by  
 amyloid beta-peptide 25-35 in middle-aged  
 rats.  
 AU Shen, Y. X. [Reprint author]; Xu, S. Y.; Wei, W.; Sun, X. X.; Liu, L. H.;  
 Yang, J.; Dong, C.  
 CS Institute of Clinical Pharmacology, Anhui Medical University, Hefei,  
 230032, China  
 shenyuxi@mail.hf.ah.cn  
 SO Journal of Pineal Research, (March, 2002) Vol. 32, No. 2, pp. 85-89.  
 print.  
 CODEN: JPRSE9. ISSN: 0742-3098.  
 DT Article  
 LA English  
 ED Entered STN: 24 Apr 2002  
 Last Updated on STN: 24 Apr 2002

L20 ANSWER 14 OF 79 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on  
 STN  
 AN 2001:98544 BIOSIS  
 DN PREV20010098544  
 TI beta-Amyloid (1-42) affects MTT reduction in astrocytes: Implications for  
 vesicular trafficking and cell functionality.  
 AU Petri, Kerokoski [Reprint author]; Hilikka, Soininen; Tuula, Pirttila  
 CS Department of Neuroscience and Neurology, University of Kuopio, 70211,  
 Kuopio, Finland  
 petri.kerokoski@ku.fi  
 SO Neurochemistry International, (February, 2001) Vol. 38, No. 2, pp.  
 127-134. print.  
 CODEN: NEUIDS. ISSN: 0197-0186.  
 DT Article  
 LA English  
 ED Entered STN: 21 Feb 2001  
 Last Updated on STN: 15 Feb 2002

L20 ANSWER 15 OF 79 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on  
 STN  
 AN 2001:92592 BIOSIS  
 DN PREV20010092592  
 TI Energy landscape theory for Alzheimer's amyloid beta-  
 peptide fibril elongation.  
 AU Massi, Francesca; Straub, John E. [Reprint author]  
 CS Department of Chemistry, Boston University, Boston, MA, 02215, USA  
 straub@bu.edu  
 SO Proteins, (February 1, 2001) Vol. 42, No. 2, pp. 217-229. print.  
 CODEN: PSFGEY. ISSN: 0887-3585.  
 DT Article  
 LA English  
 ED Entered STN: 21 Feb 2001  
 Last Updated on STN: 12 Feb 2002

- L20 ANSWER 16 OF 79 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN  
AN 2000.411441 BIOSIS  
DN PREV200000411441  
TI **peptide-induced oxidative injury.**  
AU Xiao Qiu, Wang, Rui; Tang, Xi Can [Reprint author]  
CS State Key Laboratory of Drug Research, Shanghai Institute of Materia Medica, Chinese Academy of Sciences, 294 Tai-yuan Road, Shanghai, 200031, China  
SO Journal of Neuroscience Research, (September 1, 2000) Vol. 61, No. 5, pp. 564-569. print. ISSN: 0360-4012.  
CODEN: JNREDA. ISSN: 0360-4012.  
DT Article  
LA English  
ED Entered STN: 27 Sep 2000  
Last Updated on STN: 8 Jan 2002
- L20 ANSWER 17 OF 79 CAPLUS COPYRIGHT 2007 ACS on STN  
AN 2007.452213 CAPLUS  
DN 146:516772  
TI  **$\beta$ -Sheet Structured  $\beta$ -Amyloid(1-40) Perturbs Phosphatidylcholine Model Membranes**  
AU De Planque, Maurits R. R.; Raussens, Vincent; Contera, Sonia Antoranz; Rijkers, Dirk T. S.; Liskamp, Rob M. J.; Ruysschaert, Jean-Marie; Ryan, John F.; Separovic, Frances; Watts, Anthony  
CS Biomembrane Structure Unit, Department of Biochemistry, University of Oxford, Oxford, OX1 3QU, UK  
SO Journal of Molecular Biology (2007), 368(4), .982-997  
CODEN: JMOBAK; ISSN: 0022-2836  
PB Elsevier Ltd.  
DT Journal  
LA English  
RE.CNT 90  
THERE ARE 90 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT
- L20 ANSWER 18 OF 79 CAPLUS COPYRIGHT 2007 ACS on STN  
AN 2007.152774 CAPLUS  
DN 146:160667  
TI **Chronic but not acute intracerebroventricular administration of amyloid  $\beta$ -peptide(25-35) decreases somatostatin content, adenylyl cyclase activity, somatostatin-induced inhibition of adenylyl cyclase activity, and adenylyl cyclase I levels in the rat hippocampus**  
AU Burgos-Ramos, E.; Hervas-Aguilar, A.; Puebla-Jimenez, L.; Boyano-Adanez, M. C.; Arilla-Ferreiro, E.  
CS Grupo de Neurobiología, Departamento de Bioquímica y Biología Molecular, Facultad de Medicina, Universidad de Alcalá, Alcalá de Henares, Spain  
SO Journal of Neuroscience Research (2007), 85(2), 433-442  
CODEN: JNREDA; ISSN: 0360-4012  
PB Wiley-Liss, Inc.  
DT Journal  
LA English  
RE.CNT 63  
THERE ARE 63 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT
- L20 ANSWER 19 OF 79 CAPLUS COPYRIGHT 2007 ACS on STN  
AN 2006:1205062 CAPLUS  
DN 146:161107  
TI **A $\beta$ -immunotherapy for Alzheimer's disease using mannan- *amyloid  $\beta$ -peptide* immunoconjugates**  
AU Ghochikyan, Anahit; Petrushina, Irina; Lees, Andrew; Vasilevko, Vitaly; Movsesyan, Nina; Karapetyan, Adrine; Agadjanyan, Michael G.; Cribbs, David
- H.  
CS Department of Immunology, The Institute for Molecular Medicine, Huntington Beach, CA, USA  
SO DNA and Cell Biology (2006), 25(10), 571-580  
CODEN: DCEB88; ISSN: 1044-5498  
PB Mary Ann Liebert, Inc.  
DT Journal  
LA English  
RE.CNT 65  
THERE ARE 65 CITED REFERENCES AVAILABLE FOR THIS RECORD  
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- L20 ANSWER 20 OF 79 CAPLUS COPYRIGHT 2007 ACS on STN  
AN 2006.1092969 CAPLUS  
DN 146:225706  
TI **Control of *amyloid- $\beta$* .*beta*-peptide generation by subcellular trafficking of the  $\beta$ -amyloid precursor protein and  $\beta$ -secretase**  
AU Walter, Jochen  
CS Department of Neurology, University of Bonn, Bonn, D-53127, Germany  
SO Neurodegenerative Diseases (2006), 3(4-5), 247-254  
CODEN: NDEIAG; ISSN: 1660-2854  
PB S. Karger AG  
DT Journal; General Review  
LA English  
RE.CNT 71  
THERE ARE 71 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT
- L20 ANSWER 21 OF 79 CAPLUS COPYRIGHT 2007 ACS on STN  
AN 2006.1087762 CAPLUS  
DN 146:57527  
TI **Structures of human insulin-degrading enzyme reveal a new substrate recognition mechanism**  
AU Shen, Yuequan; Joachimiak, Andrzej; Rosner, Marsha Rich; Tang, Wei-Jen  
CS Ben-May Institute for Cancer Research, The University of Chicago, Chicago, IL, 60637, USA  
SO Nature (London, United Kingdom) (2006), 443(7113), 870-874  
CODEN: NATUAS; ISSN: 0028-0836  
PB Nature Publishing Group  
DT Journal  
LA English  
RE.CNT 30  
THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD  
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- L20 ANSWER 22 OF 79 CAPLUS COPYRIGHT 2007 ACS on STN  
AN 2006:686944 CAPLUS  
DN 146:24646  
TI **The role of nitric oxide in neurodegenerative diseases and dementia**  
AU Fang, Marong; Wang, Mingwei; Qing, Renshi; Zhang, Lihong; Li, Jicheng; Yew, David T.  
CS Department of Anatomy, Medical School of Zhejiang University, Peop. Rep. China  
SO Current Trends in Neurology (2005), 1, 81-90  
CODEN: CTNUAA; ISSN: 0972-8252  
PB Research Trends  
DT Journal; General Review  
LA English  
RE.CNT 107  
THERE ARE 107 CITED REFERENCES AVAILABLE FOR THIS RECORD  
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- L20 ANSWER 23 OF 79 CAPLUS COPYRIGHT 2007 ACS on STN  
AN 2006:580664 CAPLUS  
DN 145:373539  
TI **Lipid rafts and Alzheimer's disease**  
AU Urano, Yasumi; Hamakubo, Takao



CS Department of Biochemistry, Dartmouth Medical School, Hanover, NH, 03755, USA

SO Foods & Food Ingredients Journal of Japan (2006), 211(5), 428-434

CODEN: FFIJER; ISSN: 0919-9772

PB FFI Janaru

DT Journal; General Review

LA Japanese

L20 ANSWER 24 OF 79 CAPLUS COPYRIGHT 2007 ACS on STN

AN 2006:509120 CAPLUS

DN 145:40563

TI Sex-dependent Actions of Amyloid Beta Peptides

on Hippocampal Choline Carriers of Postnatal Rats

AU Kristofikova, Z.; Richy, J.; Kozmikova, I.; Ripova, D.; Zach, P.; Klaschka, J.

CS Prague Psychiatric Center, Prague, 181 03 8, Czech Rep.

SO Neurochemical Research (2006), 31(3), 351-360

CODEN: NEREDZ; ISSN: 0364-3190

PB Springer

DT Journal

LA English

RE.CNT 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD

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L20 ANSWER 25 OF 79 CAPLUS COPYRIGHT 2007 ACS on STN

AN 2006:234583 CAPLUS

DN 144:310455

TI Anti-human amyloid beta peptide antibodies with impaired effector function for treating Alzheimer's disease, Down syndrome cerebral amyloid angiopathy, Parkinson's disease and A $\beta$  peptide-associated diseases

IN Rosenthal, Arnon; Pons, Jaume; Ho, Wei-Hsien; Grimm, Jan Markus

PA USA

SO U.S. Pat. Appl. Publ., 76 pp.

CODEN: USXXCO

DT Patent

LA English

FAN CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2006057701	A1	20060316	US 2005-194989	20050801
US 2006057702	A1	20060316	US 2005-195207	20050801
AU 2005290250	A1	20060406	AU 2005-290250	20050801
CA 2575663	A1	20060406	CA 2005-2575663	20050801
WO 2006036291	A2	20060406	WO 2005-US27295	20050801
WO 2006036291	A3	20060720		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GR, GU, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MY, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SE, SG, SK, SL, SM, SY, TJ, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GN, GO, GW, ML, MR, NE, SN, TD, TG, BM, GH, GM, KE, LS, LS, MZ, NA, SD, SL, SZ, TZ, UG, ZM, AM, AZ, BY, KG, KZ, MD, RU, T, TN				
EP 1781704	A2	20070509	EP 2005-778628	20050801
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, AU, BA, HR, MK, YU				
PRAI US 2004-592494P	P	20040730		
LA US 2005-653197P	P	20050214		

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WO 2005-US27295 W 20050801

L20 ANSWER 26 OF 79 CAPLUS COPYRIGHT 2007 ACS on STN

AN 2006:96274 CAPLUS

DN 144:485636

TI The role of an astrocytic NADPH oxidase in the neurotoxicity of amyloid beta peptides

AU Abramov, Andrey Y.; Duchen, Michael R.

CS Department of Physiology, University College London, London, WC1E 6BT, UK

SO Philosophical Transactions of the Royal Society of London, Series B: Biological Sciences (2005), 360(1464), 2309-2314

CODEN: PTRAER; ISSN: 0962-8436

PB Royal Society

DT Journal; General Review

LA English

RE.CNT 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD

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L20 ANSWER 27 OF 79 CAPLUS COPYRIGHT 2007 ACS on STN

AN 2006:38538 CAPLUS

DN 144:230912

TI Transgenic mice over-expressing human  $\beta$ -amyloid have functional nicotinic alpha 7 receptors

AU Spencer, J. P.; Weil, A.; Hill, K.; Hussain, I.; Richardson, J. C.; Cusdin, F. S.; Chen, Y. H.; Randall, A. D.

CS Neurology and GI CEDD, GlaxoSmithKline, Essex, CM19 5AW, UK

SO Neuroscience (San Diego, CA, United States) (2006), 137(3), 795-805

CODEN: NRSCDN; ISSN: 0306-4522

PB Elsevier

DT Journal

LA English

RE.CNT 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 28 OF 79 CAPLUS COPYRIGHT 2007 ACS on STN

AN 2005:903694 CAPLUS

DN 143:303830

TI The chick embryo appears as a natural model for research in beta-amyloid precursor protein processing

AU Carrodegua, J. A.; Rodolose, A.; Garza, M. V.; Sanz-Clemente, A.; Perez-Pe, R.; Lacosta, A. M.; Dominguez, L.; Monleon, I.; Sanchez-Diaz, R.; Sorribas, V.; Sarasa, M.

CS Laboratory of Neurobiology, Department of Anatomy, Embryology and Genetics, University of Zaragoza, Zaragoza, E-50013, Spain

SO Neuroscience (Oxford, United Kingdom) (2005), 134(4), 1285-1300

CODEN: NRSCDN; ISSN: 0306-4522

PB Elsevier Ltd.

DT Journal

LA English

RE.CNT 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 29 OF 79 CAPLUS COPYRIGHT 2007 ACS on STN

AN 2005:198438 CAPLUS

DN 142:424135

TI Insulin protects against amyloid beta peptide toxicity in brain mitochondria of diabetic rats

AU Oliveira, Paula I.; Santos, Maria S.; Sena, Cristina; Seica, Raquel; Moreira, Catarina R.

CS Center for Neuroscience of Coimbra, Department of Zoology, Faculty of Sciences and Technology, University of Coimbra, Coimbra, 3004-517, Port.

SO Neurobiology of Disease (2005), 18(3), 628-637

CODEN: NUDIEJ; ISSN: 0969-9961

PB Elsevier  
DT Journal  
LA English  
RE.CNT 70 THERE ARE 70 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 30 OF 79 CAPLUS COPYRIGHT 2007 ACS on STN  
AN 2004:1024659 CAPLUS  
DN 142:152749  
TI Signal transduction during amyloid-*beta*-  
peptide neurotoxicity: role in Alzheimer disease  
AU Puencelba, Rodrigo A.; Fariñas, Ginny; Scheu, Jessica; Bronfman, Miguel;  
Marzolo, Maria Paz; Inestrosa, Nibaldo C.  
CS Centro FONDAPE de Regulación Celular y Patología "Joaquín Luco", MIFAB,  
Facultad de Ciencias Biológicas, Pontificia Universidad Católica de Chile,  
Santiago, Chile  
SO Brain Research Reviews (2004), 47(1-3), 275-289  
CODEN: BRERD2; ISSN: 0165-0173  
PB Elsevier B.V.  
DT Journal; General Review  
LA English  
RE.CNT 163 THERE ARE 163 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 31 OF 79 CAPLUS COPYRIGHT 2007 ACS on STN  
AN 2004:100490 CAPLUS  
DN 140:124840  
TI Methods for detecting parenchymal plaques in vivo  
IN Poduelo, Joseph F.; Curran, Geoffrey L.; Wengenack, Thomas M.  
PA USA  
SO U.S. Pat. Appl. Publ., 25 pp., Cont.-in-part of U.S. Ser. No. 542,537,  
abandoned.  
CODEN: USXXCO  
DT Patent  
LA English  
FAN CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2004022736	A1	20040205	US 2003-351777	20030127
PRAI US 2000-542537	B2	20000404		
US 2002-427821P	P	20021120		

L20 ANSWER 32 OF 79 CAPLUS COPYRIGHT 2007 ACS on STN  
AN 2003:826200 CAPLUS  
DN 139:360646  
TI Influence of Hydrophobic Teflon Particles on the Structure of  
Amyloid-*beta*-Peptide  
AU Giacomelli, Carla E.; Norde, Willem  
CS INPIQ Departamento de Fisicoquímica, Facultad de Ciencias Químicas,  
Universidad Nacional de Córdoba, Córdoba, 5000, Argent.  
SO Biomacromolecules (2003), 4(6), 1719-1726  
CODEN: BOMAF6; ISSN: 1525-7797  
PB American Chemical Society  
DT Journal  
LA English  
RE.CNT 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD  
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L20 ANSWER 33 OF 79 CAPLUS COPYRIGHT 2007 ACS on STN  
AN 2003:701932 CAPLUS  
DN 139:301297  
TI Stereoselective Interactions of Peptide Inhibitors with the *beta*-  
Amyloid Peptide  
AU Chalfour, Robert J.; McLaughlin, Richard W.; Lavoie, Louis; Morissette,

Celine; Tremblay, Nadine; Boule, Marie; Sarazin, Philippe; Stea, Dino;  
Lacombe, Diane; Tremblay, Patrick; Gervais, Francine  
CS Neurochem Inc., Saint-Laurent, QC, H4S 2A1, Can.  
SO Journal of Biological Chemistry (2003), 278(37), 34874-34881  
CODEN: JBCHAJ; ISSN: 0021-9258  
PB American Society for Biochemistry and Molecular Biology  
DT Journal  
LA English  
RE.CNT 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD  
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L20 ANSWER 34 OF 79 CAPLUS COPYRIGHT 2007 ACS on STN  
AN 2003:346501 CAPLUS  
DN 139:114944  
TI Cholesterol Distribution in the Golgi Complex of DITNC1 Astrocytes Is  
Differentially Altered by Fresh and Aged Amyloid-*beta*  
--Peptide-(1-42)  
AU Igbavboa, Urule; Pidcock, Justine M.; Johnson, Leslie N. A.; Malo, Todd  
M.; Studniski, Ann E.; Yu, Su; Sun, Grace Y.; Wood, W. Gibson  
CS Veterans Affairs Medical Center and the Department of Pharmacology,  
Education and Clinical Center, Geriatric Research, University of Minnesota  
School of Medicine, Minneapolis, MN, 55417, USA  
SO Journal of Biological Chemistry (2003), 278(19), 17150-17157  
CODEN: JBCHAJ; ISSN: 0021-9258  
PB American Society for Biochemistry and Molecular Biology  
DT Journal  
LA English  
RE.CNT 61 THERE ARE 61 CITED REFERENCES AVAILABLE FOR THIS RECORD  
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L20 ANSWER 35 OF 79 CAPLUS COPYRIGHT 2007 ACS on STN  
AN 2002:893187 CAPLUS  
DN 138:269080  
TI Alzheimer's disease and the basal forebrain cholinergic system: relations  
to *beta*-amyloid peptides, cognition, and  
treatment strategies  
AU Auld, Daniel S.; Kornecook, Tom J.; Bastianetto, Stephanie; Quirion, Remi  
CS Douglas Hospital Research Centre, Verdun, QC, H4H 1R3, Can.  
SO Progress in Neurobiology (Oxford, United Kingdom) (2002), 68(3), 209-245  
CODEN: PGNBAS; ISSN: 0301-0082  
PB Elsevier Science Ltd.  
DT Journal; General Review  
LA English  
RE.CNT 504 THERE ARE 504 CITED REFERENCES AVAILABLE FOR THIS RECORD  
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L20 ANSWER 36 OF 79 CAPLUS COPYRIGHT 2007 ACS on STN  
AN 2002:508402 CAPLUS  
DN 137:212549  
TI Charge states rather than propensity for  $\beta$ -structure determine  
enhanced fibrillogenesis in wild-type Alzheimer's *beta*-  
amyloid peptide compared to E22Q Dutch mutant  
amyloid peptide  
AU Massi, Francesca; Klimov, D.; Thirumalai, D.; Straub, John E.  
CS Department of Chemistry, Boston University, Boston, MA, 02215, USA  
SO Protein Science (2002), 11(7), 1639-1647  
CODEN: PROSCI; ISSN: 0961-8368  
PB Cold Spring Harbor Laboratory Press  
DT Journal  
LA English  
RE.CNT 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD  
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L20 ANSWER 37 OF 79 CAPLUS COPYRIGHT 2007 ACS on STN  
AN 2002:267661 CAPLUS

DN 136:396235  
 TI The protective effects of melatonin from oxidative damage induced by  
 amyloid **beta-peptide** 25-35 in middle-aged rats  
 AU Shen, Y. X.; Xu, S. Y.; Wei, W.; Sun, X. X.; Liu, L. H.; Yang, J.; Dong,  
 C.  
 CS Institute of Clinical Pharmacology, Anhui Medical University, Hefei,  
 230032, Peop. Rep. China  
 SO Journal of Pineal Research (2002), 32(2), 85-89  
 CODEN: JPRS9J; ISSN: 0742-3098  
 PB Blackwell Munksgaard  
 DT Journal  
 LA English  
 RE.CNT 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD  
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L20 ANSWER 38 OF 79 CAPLUS COPYRIGHT 2007 ACS on STN  
 AN 202:228581 CAPLUS  
 DN 137:153285  
 TI Suppressed expression of nicotinic acetylcholine receptors by nanomolar  
**beta-amyloid peptides** in PC12 cells  
 AU Guan, Z.-Z.; Miao, H.; Tian, J.-Y.; Unger, C.; Nordberg, A.; Zhang, X.  
 CS Division of Molecular Neuropharmacology, Department of Clinical, Huddinge  
 University Hospital, Huddinge, Sweden  
 SO Journal of Neural Transmission (2001), 108(12), 1417-1433  
 CODEN: JNTRFJ; ISSN: 1435-1463  
 PB Springer-Verlag Wien  
 DT Journal  
 LA English  
 RE.CNT 54 THERE ARE 54 CITED REFERENCES AVAILABLE FOR THIS RECORD  
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L20 ANSWER 39 OF 79 CAPLUS COPYRIGHT 2007 ACS on STN  
 AN 201:557510 CAPLUS  
 DN 135:339064  
 TI **Amyloid beta peptide** 1-40 and the  
 function of rat hippocampal hemicholinium-3 sensitive choline  
 carriers: effects of a proteolytic degradation in vitro  
 AU Kristofikova, Zdena; Tejkalova, Hana; Kraschka, Jan  
 CS Prague Psychiatric Centre, Prague, 18103/8, Czech Rep.  
 SO Neurochemical Research (2001), 26(3), 203-212  
 CODEN: NERED2; ISSN: 0364-3190  
 PB Kluwer Academic/Plenum Publishers  
 DT Journal  
 LA English  
 RE.CNT 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD  
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L20 ANSWER 40 OF 79 CAPLUS COPYRIGHT 2007 ACS on STN  
 AN 201:80286 CAPLUS  
 DN 134:264528  
 TI **Beta-Amyloid** (1-42) affects WTT reduction in astrocytes: implications  
 for vesicular trafficking and cell functionality  
 AU Kerokoski, Petri; Soininen, Hilka; Pirttila, Tuula  
 CS Department of Neuroscience and Neurology, University of Kuopio, Kuopio,  
 70211, Finland  
 SO Neurochemistry International (2001), 38(2), 127-134  
 CODEN: NEUIDS; ISSN: 0197-0186  
 PB Elsevier Science Ltd.  
 DT Journal  
 LA English  
 RE.CNT 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD  
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L20 ANSWER 41 OF 79 CAPLUS COPYRIGHT 2007 ACS on STN

AN 201:59884 CAPLUS  
 DN 134:250689  
 TI Energy landscape theory for Alzheimer's **amyloid beta**  
**-peptide** fibril elongation  
 AU Massi, Francesca; Straub, John E.  
 CS Department of Chemistry, Boston University, Boston, MA, 02215, USA  
 SO Proteins: Structure, Function, and Genetics (2001), 42(2), 217-229  
 CODEN: PSFGEY; ISSN: 0887-3585  
 PB Wiley-Liss, Inc.  
 DT Journal  
 LA English  
 RE.CNT 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD  
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L20 ANSWER 42 OF 79 CAPLUS COPYRIGHT 2007 ACS on STN  
 AN 200:655982 CAPLUS  
 DN 133:329417  
 TI Huperzine A and tacrine attenuate **beta-amyloid**  
**peptide**-induced oxidative injury  
 AU Xiao, Xiao Qiu; Wang, Rui; Tang, Xi Can  
 CS State Key Laboratory of Drug Research, Shanghai Institute of Materia  
 Medica, Chinese Academy of Sciences, Shanghai, 200031, Peop. Rep. China  
 SO Journal of Neuroscience Research (2000), 61(5), 564-569  
 CODEN: JNRRED; ISSN: 0360-4012  
 PB Wiley-Liss, Inc.  
 DT Journal  
 LA English  
 RE.CNT 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD  
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L20 ANSWER 43 OF 79 CAPLUS COPYRIGHT 2007 ACS on STN  
 AN 200:569427 CAPLUS  
 DN 134:98483  
 TI **Beta-Amyloid** augments platelet aggregation. Reduced activity of familial  
 angiotensin-converting enzyme mutants  
 AU Wolosin, B.; Maheshwari, S.; Jones, C.; Dukoff, R.; Wallace, W.; Racchi,  
 M.; Nagula, S.; Shulman, N. R.; Sunderland, T.; Bush, A.  
 CS Section on Geriatric Psychiatry, NIMH, Bethesda, MD, 20892, USA  
 SO Molecular Psychiatry (1998), 3(6), 500-507  
 CODEN: MOPSFQ; ISSN: 1359-4184  
 PB Stockton Press  
 DT Journal  
 LA English  
 RE.CNT 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD  
 ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 44 OF 79 CAPLUS COPYRIGHT 2007 ACS on STN  
 AN 200:156752 CAPLUS  
 DN 132:277675  
 TI **Beta-Amyloid** 1-42 binds to  $\alpha 7$  nicotinic acetylcholine receptor with  
 high affinity: implications for Alzheimer's disease pathology  
 AU Wang, Houan-Yan; Lee, Daniel H. S.; D'Andrea, Michael R.; Peterson, Per A.;  
 Shank, Richard P.; Reitz, Allen B.  
 CS R. W. Johnson Pharmaceutical Research Institute, Spring House, PA,  
 19477-0776, USA  
 SO Journal of Biological Chemistry (2000), 275(8), 5626-5632  
 CODEN: JBCHA3; ISSN: 0021-9258  
 PB American Society for Biochemistry and Molecular Biology  
 DT Journal  
 LA English  
 RE.CNT 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD  
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L20 ANSWER 45 OF 79 CAPLUS COPYRIGHT 2007 ACS on STN

AN 1997:774254 CAPLUS  
DN 128:71065  
TI 1 $\beta$ -Estradiol attenuates oxidative impairment of synaptic  
NA+/K+-ATPase activity, glucose transport, and glutamate transport induced  
by amyloid  $\beta$ -peptide and iron  
AU Keller, Jeffrey N.; Gernsey, Ariane; Begley, James G.; Mattson, Mark P.  
CS Sanders-Brown Research Center on Aging, University of Kentucky, Lexington,  
KY, USA  
SO Journal of Neuroscience Research (1997), 50(4), 522-530  
CODEN: JNREDA; ISSN: 0360-4012  
PB Wiley-Liss, Inc.  
DT Journal  
LA English  
RE.CNT 81 THERE ARE 81 CITED REFERENCES AVAILABLE FOR THIS RECORD  
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L20 ANSWER 46 OF 79 CAPLUS COPYRIGHT 2007 ACS on STN  
AN 1997:700729 CAPLUS  
DN 127:329848  
TI  $\beta$ -Amyloid-induced cerebrovascular endothelial dysfunction  
AU Thomas, Tom; McIndon, Chris; Sutton, E. Truitt; Thomas, George  
CS Departments of Psychiatry and Physiology, College of Medicine, Univ. South  
Florida, Tampa, FL, 33613, USA  
SO Annals of the New York Academy of Sciences (1997), 826(Cerebrovascular  
Pathology in Alzheimer's Disease), 447-451  
CODEN: ANVA99; ISSN: 0077-8923  
PB New York Academy of Sciences  
DT Journal  
LA English  
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L20 ANSWER 47 OF 79 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights  
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AN 2007178627 EMBASE  
TI  $\beta$ -Sheet Structured  $\beta$ -Amyloid(1-40) Perturbs Phosphatidylcholine  
Model Membranes  
AU de Planque M.R.; Raussens V.; Contera S.A.; Rijkers D.T.S.; Liskamp  
R.M.J.; Ruyschaert J.-M.; Ryan J.F.; Separovic F.; Watts A.  
CS M.R. de Planque, Biomembrane Structure Unit, Department of Biochemistry,  
University of Oxford, South Parks Road, Oxford, OX1 3QU, United Kingdom.  
m.deplanque@physics.ox.ac.uk  
SO Journal of Molecular Biology, (11 May 2007) Vol. 368, No. 4, pp. 982-997.  
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ISSN: 0022-2836 CODEN: JMOBAK  
PUI S 0022-2836(07)00255-0  
CY United Kingdom  
DT Journal; Article  
FS 029 Clinical Biochemistry  
LA English  
SL English  
ED Entered STN: 1 May 2007  
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expression and caspase-3 activation. A crucial role for the redox state of  
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Navarra P.; Giardina B.; Pozzoli G.

CS G. Pozzoli, Institute of Pharmacology, Catholic University School of  
Medicine, Largo F. Vito 1, 00168 Rome, Italy. giacompozzi@rm.unicatt.it  
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ISSN: 0197-0186 CODEN: NEUIDS  
PUI S 0197-0186(06)00149-5  
CY United Kingdom  
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FS 005 General Pathology and Pathological Anatomy  
008 Neurology and Neurosurgery  
029 Clinical Biochemistry  
LA English  
SL English  
ED Entered STN: 29 Aug 2006  
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L20 ANSWER 49 OF 79 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights  
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nicotinic alpha 7 receptors  
AU Spencer J.P.; Weil A.; Hill K.; Hussain I.; Richardson J.C.; Cusdin F.S.;  
Chen Y.H.; Randall A.D.  
CS J.P. Spencer, Neurology and GI CEDD, GlaxoSmithKline, Harlow, Essex CM19  
5AW, United Kingdom. jon\_p\_spencer@gsk.com  
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ISSN: 0306-4522 CODEN: NRSQDN  
PUI S 0306-4522(05)01150-4  
CY United Kingdom  
DT Journal; Article  
FS 008 Neurology and Neurosurgery  
029 Clinical Biochemistry  
LA English  
SL English  
ED Entered STN: 26 Jan 2006  
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L20 ANSWER 50 OF 79 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights  
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TI Assessment of the bioactivity of antibodies against  $\beta$ -amyloid peptide  
in vitro and in vivo.  
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Kuehnle K.; Wollmer M.A.; Nitsch R.M.  
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August Forel Strasse 1, CH-8008 Zurich, Switzerland. mohajeri@li.unizh.ch  
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ISSN: 1660-2854 CODEN: NDEIA6  
CY Switzerland  
DT Journal; Article  
FS 008 Neurology and Neurosurgery  
026 Immunology, Serology and Transplantation  
037 Drug Literature Index  
LA English  
SL English  
ED Entered STN: 9 Dec 2004  
Last Updated on STN: 9 Dec 2004

L20 ANSWER 51 OF 79 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights  
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tissue culture.

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 CS J. Gotz, Division of Psychiatry Research, University of Zurich, August  
 Forel Strasse 1, 8008 Zurich, Switzerland. goetz@bli.unizh.ch  
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 ISSN: 0021-9258 CODEN: JBCHA3  
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 DT Journal, Article  
 FS 005 General Pathology and Pathological Anatomy  
 006 Neurology and Neurosurgery  
 029 Clinical Biochemistry  
 LA English  
 SL English  
 ED Entered STN: 6 Nov 2003  
 Last Updated on STN: 6 Nov 2003

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 treatment strategies.  
 AU Auld D.S.; Kornecook T.J.; Bastianetto S.; Quirion R.  
 CS R. Quirion, Douglas Hospital Research Centre, 6875 Blvd. Lasalle, Verdun,  
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 ISSN: 0304-0082 CODEN: PGNBAA  
 PUI S 0301-0082(02)00079-5  
 CY United Kingdom  
 DT Journal, General Review  
 FS 008 Neurology and Neurosurgery  
 029 Clinical Biochemistry  
 030 Pharmacology  
 037 Drug Literature Index  
 038 Adverse Reactions Titles  
 LA English  
 SL English  
 ED Entered STN: 12 Dec 2002  
 Last Updated on STN: 12 Dec 2002

L20 ANSWER 53 OF 79 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights  
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 amyloid-*beta* peptides, induces neuronal  
 apoptosis.  
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 CS J.-N. Octave, Université Catholique de Louvain, FARL/ UCL 54 10, Av  
 Hippocrate 54, B-1200 Brussels, Belgium. octave@nchm.ucl.ac.be  
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 ISSN: 0021-9258 CODEN: JBCHA3  
 United States  
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 DT 005 General Pathology and Pathological Anatomy  
 FS 008 Neurology and Neurosurgery  
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 SL English  
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 AU Massi F.; Klimov D.; Thirumalai D.; Straub J.E.  
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 Avenue, Boston, MA 02215, United States. straube@bu.edu  
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 FS 029 Clinical Biochemistry  
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 ED Entered STN: 11 Jul 2002  
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L20 ANSWER 56 OF 79 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights  
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 CS J.E. Straub, Department of Chemistry, Boston University, Boston, MA 02215,  
 United States. straube@bu.edu  
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 Refs: 36  
 ISSN: 0887-3585 CODEN: PSFGEY  
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 SL English  
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Last Updated on STN: 25 Jan 2001

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AN 2001010030 EMBASE

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AU Kerokoski P.; Soininen H.; Pirttilä T.

CS P. Kerokoski, Department of Neuroscience/Neurology, University of Kuopio, PO Box 1627, 70211 Kuopio, Finland. petri.kerokoski@uku.fi

SO Neurochemistry International, (1 Feb 2001) Vol. 38, No. 2, pp. 127-134.

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ISBN: 0197-0186 CODEN: NEUIDS

PUI S 0197-0186(00)00071-1

CY United Kingdom

DT Journal; Article

FS 008 Neurology and Neurosurgery

029 Clinical Biochemistry

LA English

SL English

ED Entered STN: 1 Feb 2001

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AN 200314251 EMBASE

TI Hupezzine A and tacrine attenuate  $\beta$ -amyloid peptide-induced oxidative injury.

AU Xiao Qiu Xiao; Wang R.; Xi Can Tang

CS Prof. X.C. Tang, State Key Lab. of Drug Research, Shanghai Institute of Materia Medica, Chinese Academy of Sciences, 294 Tai-yuan Road, Shanghai 200031, China. xctang@mail.shnc.ac.cn

SO Journal of Neuroscience Research, (1 Sep 2000) Vol. 61, No. 5, pp. 564-569.

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ISBN: 0360-4012 CODEN: JNREDK

CY United States

DT Journal; Article

FS 008 Neurology and Neurosurgery

037 Drug Literature Index

LA English

SL English

ED Entered STN: 21 Sep 2000

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AN 2007:108272 TOXCENTER

CP Copyright (c) 2007 The Thomson Corporation

DN PREV200700315208

TI Abeta Peptide and Alzheimer's Disease: Celebrating a Century of Research

AU Barrow, CJ [Editor]; Small, DH [Editor]

CS Ocean Nutr Canada, Dartmouth, NS, Canada

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LA English

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AN 2007:66414 TOXCENTER

CP Copyright 2007 ACS

DN CA1461225706T

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AU Walter, Jochen

CS Department of Neurology, University of Bonn, Bonn, D-53127, Germany.

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CODEN: NDETAG. ISSN: 1660-2854.

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ANSWER 61 OF 79 TOXCENTER COPYRIGHT 2007 ACS on STN

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DN CA14609160667J

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AU Burgos-Ramos, E.; Hervás-Aguilar, A.; Puebla-Jimenez, L.; Boyano-Adanez, M. C.; Arilla-Ferreiro, E.

CS Grupo de Neurobioquímica, Departamento de Bioquímica y Biología Molecular, Facultad de Medicina, Universidad de Alcalá, Alcalá de Henares, Spain.

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AN 2007:40872 TOXCENTER

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DN CA14609161107P

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AU Ghochikyan, Anahit; Petrushina, Irina; Lees, Andrew; Vasilevko, Vitaly; Movsesyan, Nina; Karapetyan, Adrine; Agadjanyan, Michael G.; Cribbs, David H.

CS Department of Immunology, The Institute for Molecular Medicine, Huntington Beach, CA, USA.

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Last Updated on STN: 5 Jun 2007
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TI Anti-human amyloid .beta. peptide antibodies  
with impaired effector function for treating Alzheimer's  
disease, Down syndrome cerebral amyloid angiopathy, Parkinson's disease  
and Aβ peptide-associated diseases  
AU Rosenthal, Arnon; Pons, Jaume; Ho, Wei-Hsien; Grimm, Jan Markus  
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CODEN: USXXCO.
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Sciences and Technology, University of Coimbra, Coimbra, 3004-517, Port..  
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LA English  
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AN 2004:271381 TOXCENTER  
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peptide neurotoxicity: role in Alzheimer disease  
FUentealba, Rodrigo A.; Farias, Ginny; Scheu, Jessica; Bronfman, Miguel;  
Marzolo, Maria Paz; Inestrosa, Nibaldo C.  
CS Centro FONDAP de Regulacion Celular y Patologia "Joaquin Luco", MIFAB,  
Facultad de Ciencias Biologicas, Pontificia Universidad Catolica de Chile,  
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Last Updated on STN: 22 Feb 2005
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LA English  
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to .beta.-amyloid peptides, cognition, and  
treatment strategies  
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CY CANADA

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LA English

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